VOLUME 65 • APRIL 1957 • NUMBER 4

A.M.A. ARCHIVES OF

OTOLARYNGOLOGY

Vocal Rehabilitation of Paralytic Dysphonia
Godfrey E. Arnold

The Frontonasal Angle in Cosmetic Rhinoplasty
Benito B. Rish

Dorsal Sympathectomy in Labyrinthine Disease
M. Stuart Strong

Congenital Atresia of the Middle and External Ear
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ARCHIVES OF OTOLARYNGOLOGY

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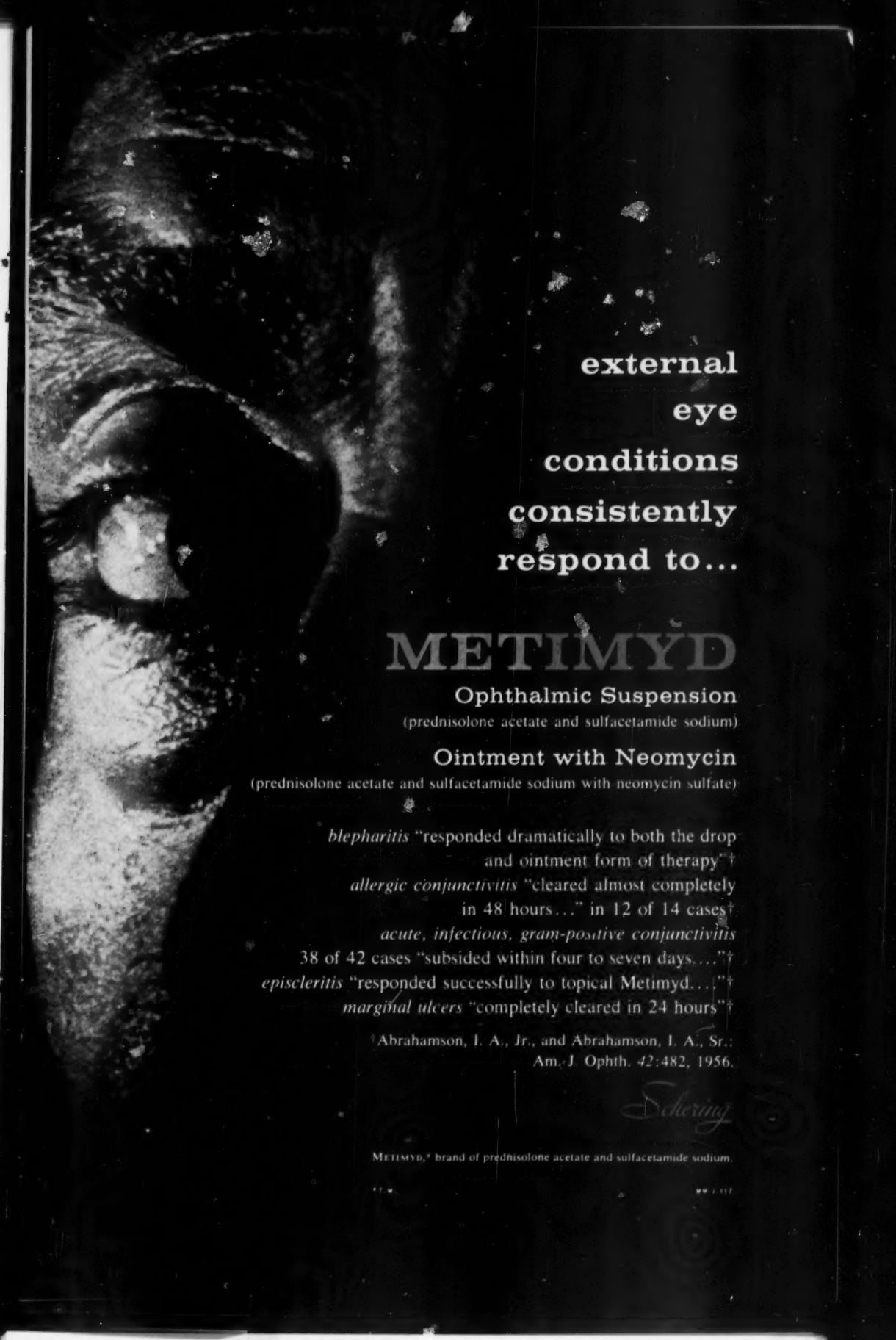

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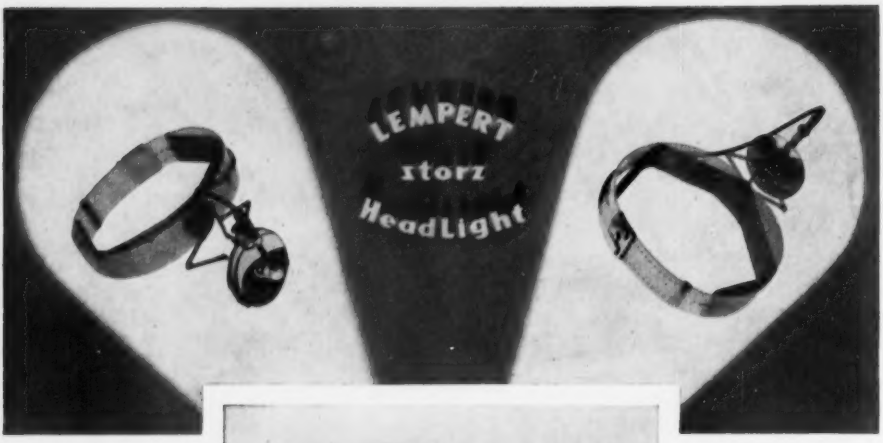

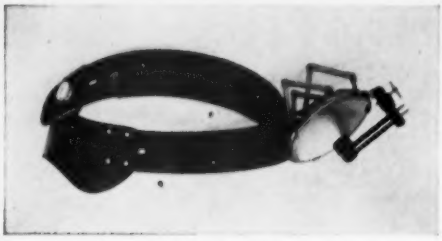

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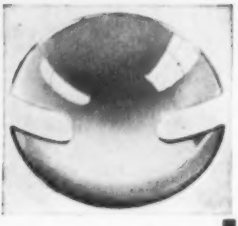

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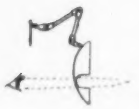

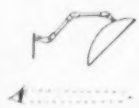

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Vocal Rehabilitation of Paralytic Dysphonia

III. Present Concepts of Laryngeal Paralysis

GODFREY E. ARNOLD, M.D., New York

Introduction

Perusing the tremendous literature on paralysis of the larynx, one cannot help wondering why it required the untiring efforts of hundreds of scholars to unravel the mysterious problems connected with paralyzed vocal cords. During the last hundred years periods of diligent experimentation were followed by innumerable and, at times, rather temperamental argumentations. After the discussions had calmed, resignation reigned for a time and many authors preferred an attitude of guarded neutrality when dealing with the controversial questions of laryngoneurology. Lately more general agreement seems to have been reached and some authors propound the final explanation of all pertinent problems with definite affirmance.

Contrary to other clinical or therapeutic topics, the older bibliography on laryngeal paralysis is not necessarily limited by obsolescence. Whereas the advent of new drugs or of bacteriologic advances has radically altered most aspects of medical practice, the problems of normal and diseased laryngeal innervation remained the same. They represent discussions of clinical observations, of experimental findings, and of their interpretations. Moreover, most of the older publications contain many detailed observations and clinical studies which should not be left to oblivion. At least these previous efforts offer much help in avoiding certain experimental mistakes, as has been remarked also by Murtagh and Campbell. It seems that some of the previous misunderstandings were due to the misapplication of animal experimentation to human pathology. In general, the following conclusions can be drawn from the gross of the older papers.

Accepted for publication May 25, 1956.

From the Department of Otolaryngology (John F. Daly, M.D., Director), New York University Post-Graduate Medical School.

Some Problems Encountered in Previous Research

1. The human larynx is a very complicated organ which has developed into a specifically human structure, capable of producing the great variety of human voice sounds. It is one of the organs which show the widest individual variations of functional perfection, ranging from the vague states of constitutional weakness to the vocal virtuosity of the admired singer.

2. There are important anatomic and physiologic differences between the human larynx and that of animals. They increase with the distance of phylogenetic affinity (de Beule, Frischbier, Negus, Pressman and Kelemen, A. Réthi, Stupka). It is therefore impossible to establish rules of human laryngeal innervation solely because such rules have been found to be true in animals (Broeckaert, Clerf and Baltzell,

Elze, Gleitsmann, Hofer and Jeschek, Katzenstein).

Horses have a double interarytenoid muscle with a median tendinous raphe between the two muscle portions (King and Gregg). The same was found in the dog by Réthi. Even the bulbar nuclei appear to be arranged in a different manner from that in man. Broeckaert, de Beule, and others believed that in rabbits the dorsal nucleus of the vagus was the seat of motor laryngeal innervation. Provided that this finding is not a simple error, it would mean a basic difference from the human relationship between the recurrent nerve and its motor ventral nucleus ambiguus of the vagus. Dogs and rabbits usually have three laryngeal nerves, namely, the superior, inferior, and Exner's median laryngeal nerve. This is a branch of the pharyngeal plexus. It assists the superior laryngeal nerve in the innervation of the cricothyroid muscle (de Beule, Katzenstein, Lasagna, Roberto, Tassi). According to Elze, the inferior laryngeal nerve is not recurrent in the llama. A human nonrecurrent inferior laryngeal was found by Pereira, Lahey and Hoover, and R. G. Williams.

3. Individual variations of the laryngeal nerve supply are very common in man. The course of the laryngeal nerves, their divisions, ramifications, anastomoses, distributions, relations to the accompanying blood vessels, and their bilateral symmetry are said to vary as much as do the fingerprints (A. Réthi). These individual differences provide ready explanations for apparent exceptions of the behavior of paralyzed vocal cords. Pertinent observations were made by Armstrong and Hinton, Berendes, Fabbi, Grabower, Graceva, Hjelmman, Hofer and Jeschek, Imperatori, Kaspar, King and Gregg, Kluyskens, Kressner, Luchsinger, Marschik, Meurman, L. F. Morrison, Negus, Rustad and Morrison, Tarneaud, P. H. Vogel, Wessely, etc. Thanks are due to these authors, for they help us to explain some of the unusual clinical findings which cannot otherwise be explained.

4. Physiologic experiments on the vocal organs of animals present many technical difficulties. Apart from the anatomic and physiologic differences mentioned above, the following sources of error should be borne in mind: the small dimensions of the larynx in the usual laboratory animals; ambiguous results of electrical stimulations; the problematic nature of any attempt at isolating a particular physiologic function from the complicated interplay of all laryngeal structures, and pharmacologic influences of anesthetic agents on the laryngeal movements.

Since electric currents follow the pathways of least resistance, and since they also spread over adjacent conductive structures, it can be very difficult to demonstrate isolated electrical reactions. Many times positive results have been simulated by the mere spread of currents to such structures which were supposedly not under consideration (Stupka). Similar caution is necessary with regard to partial dissections for the isolation of certain functions. It is often unavoidable to damage other structures which seem to be a component part of the vocal mechanism (Klemperer). Many of the older experiments lead to faulty conclusions because they did not clearly isolate the particular function under study. Semon and Horsley, A. Réthi, Stupka, and others have shown how the type and depth of general anesthesia influences the position and the movements of normal and paralytic vocal cords of animals. Therefore, the technique of anesthesia adds to the variances of the experimental procedure (Grabower).

5. It is astonishing how often the same findings were reported by various authors. Yet, many of them frequently arrived at different conclusions, using the same observations to prove divergent assumptions. In fact, almost every statement on laryngeal innervation may be either corroborated or rejected by quoting previous opinions. One cannot but admire the diligence and ingenuity of the older investigators and their experiments. Many of the observations which are so abundantly reported in the pertinent literature appear perfectly valid to this day. Rather, it was the problem of correct interpretation of the findings which lead many astray. This had repeatedly been said also with reference to Sir Felix Semon, whose arduous work never failed to elicit admiration, but whose dogmatic deductions and "laws" aroused increasing contradiction.

6. Finally, a widespread confusion of terminology precipitated many superfluous litigations. Although careful attempts at definition date back as far as 1893 (R. Wagner), no one could be sure that he knew

exactly what another author meant when speaking of the clinically important vocal cord positions (cf. the Semon-Grossmann controversy). Especially the controversial, because nonexistent, "cadaveric" position caused much confusion. Broeckaert regarded the "cadaveric" position as identical with the paramedian position and clearly differentiated it from the intermediate position which is midway between adduction and abduction. Others located the "cadaveric" position in the intermediary line. As is now generally understood, the vocal cords may be found in a great variety of positions after death, including firm closure (Lauffs). It had, therefore, been proposed innumerable times to relinquish this term.

Changing Concepts of Laryngeal Paralysis

Although any attempt at historical classification always reflects the writer's subjective impression, the milestones of neurolaryngologic research may be presented in the following manner.

The Period 1854-1880.—This is the era of early laryngology, which began with Garcia's invention of the laryngeal mirror. In rapid succession there appeared the first treatises on clinical laryngology, such as those by Türck, and Czermak, with the first attempts at clinical classification of laryngeal disease. Together with the physiologists the early pioneers drew the laryngoscopic images of paralytic conditions, deducing theoretically from the then known actions of the laryngeal muscles what their paralyses should look like (Türck, 1860; Gerhardt, 1863).

Most of these beautiful illustrations still depict what we see nowadays in our clinics. Some, however, originated the traditional errors which subsequently have been copied from book to book and continue to bewilder the novice. One of these errors is the laryngoscopic picture of isolated paralysis of the superior laryngeal nerve or of the corresponding cricothyroid muscle. Since this condition is rare and even less frequently recognized (Mainzer), there seems to be little agreement on its true laryngoscopic appearance. Hence, the textbooks present quite divergent drawings. These have only one common feature: the famous wavy or even serpentine cords. Although a cord without tension cannot possibly look like a snake, and although this appearance does not yet seem to have been visualized, these obsolete illustrations continue to contradict the accepted verbal descriptions: "The mirror appearance of the cords is stated to be wavy or lacking in normal tension with an oblique glottis" (W. W. Morrison).

The Period 1880-1920.—After Rosenbach had described in 1880 his observation of the greater susceptibility of the abductor muscle to partial paralysis, Semon began to publish, in 1881, his papers proclaiming and defending what became known as Semon's law (or the law of Rosenbach and Semon, as the German authors preferred to call it). This law tried to explain the sequence of paralytic conditions of the vocal cords. Accordingly, four stages of laryngeal paralysis were distinguished: (1) isolated abductor paralysis, (2) midline position as a result of spastic contracture of the adductors, (3) cadaveric position in the stage of complete paralysis of all muscles innervated by the recurrent nerve, and (4) compensation of the paralytic condition by the crossing over the midline of the healthy cord. During the subsequent years a great deal of work was devoted to the question whether Semon was right, or his opponents, and much paper was filled with bitter controversy.

The main opponents were Wagner (1890) and Grossmann (1897). They maintained that the median or paramedian position of the paralyzed vocal cord was due to the remaining action of the superior laryngeal nerve and its cricothyroid muscle. Intermediary position would appear only after both the inferior and the superior laryngeal nerves were paralyzed. Although Grossmann corroborated his views with experimental evidence, he was supported by only a few others, mostly by Krause and to some extent by Broeckaert. The large majority of investigators, including du Bois-Reymond, Katzenstein, Klemperer, Kuttner, Onodi, and others, remained on Semon's side. Much time has passed since then, and it is interesting to note that the essence of Grossmann's opinion has later been revived

by many authors. It now represents the widely accepted interpretation of laryngeal paralysis.

The Period 1920-1950.—As soon as scientific activity was resumed after World War I (Fabry, 1921), many important contributions began to reevaluate the complicated problems of laryngeal paralysis. Much had been learned from the isolated and associated paralytic conditions due to war injuries which could no longer be reconciled with Semon's law.

The German Otolaryngological Society entrusted in 1921 a special commission (composed of Neumayer, Stupka, Klestadt, and Spiess) with the study and formulation of the unsolved problems of laryngeal paralysis. A detailed questionnaire was published in 1922, and future investigators were invited to proceed accordingly. This was the beginning of the official skepticism in, and the ultimate rejection of, the universal validity of Rosenbach and Semon's law.

In 1923 New explained that complete paralysis of the recurrent laryngeal nerve produced paramedian position of the vocal cord, while the intermediate position was due to the combined and complete paralysis of both the superior and inferior laryngeal nerves. Leichsenring and Hegener (1924) were the first to investigate the problem experimentally on human patients. On the grounds of these and similar observations, many authors began to reject completely any validity of Semon's law. Others now prefer to speak of Semon's rule when referring to the accepted greater vulnerability of the abductor function (Clerf, Jackson and Jackson, King and Gregg, Klestadt, O. Körner, Lederer, P. and L. G. Mangabeira-Albernaz, Myerson, Parkinson, Piquet, A. Réthi, Spiess, Tarneaud, Thomson and Negus, A. F. Williams).

Then followed the profound monographs in the "Handbuch" by Denker and Kahler, 1925-1929 (Elze, v. Skramlik, Amersbach), with their encyclopedic reviews of the accumulated information. New and Childrey (1932), Lemere (1933-1934), Hofer and Jeschek (1940), Clerf and Suehs (1941), Thomson and Negus (1947), Jeschek (1953), and others added new evidence in favor of the original theory by Wagner and Grossmann: It is the cricothyroid muscle which holds the paralyzed cord in the median or paramedian position, whereas intermediate position is the result of combined paralysis of both the superior and inferior laryngeal nerves.

Those favoring the modern distinction of complete and combined types of paralysis include Ballenger, Callaghan, Clerf and Baltzell, Della Cioppa, Fischer, Font, Freedman, Fritzsche, Hörbst, Holzer, Kecht, King and Gregg, Kressner, Looper, Lore, Luchsinger, Meurman, W. W. Morrison, Musgrove, Oltersdorf, Pressman and Kelemen, A. Réthi, Rüedi, Titche, Thomson and Negus. Among the opponents of this explanation are such authors as Portmann, who interprets the paramedian position as a recent paralysis, while intermediate position would reflect a paralysis of old standing; others adhere to Semon's concept that the paramedian position means partial paralysis, while the intermediate position indicates complete recurrent paralysis (Glas, Habermann, Krejci, L. F. Morrison, Piquet, K. Vogel).

The year 1950 represents a new turning point. Since that year Husson has arduously propagated his new concept of vocal cord vibration. According to his neurochronaxic theory of phonation, each vocal cord vibration is activated by a synchronous and discrete nervous impulse. Generated within the cerebral cortex, these impulses would also reflect the cerebral modification of all vocal functions, including the manifold influences of the subcortical, bulbar, and cerebellar centers and pathways on the limitless differentiations of vocal expression. Whether or not this revolutionary theory can successfully explain the complicated phenomena of human vocal expression (Panconcelli-Calzia) need not be discussed here. In any case, it is certain that these new electrophysiologic studies of vocal phenomena have already given a tremendous impetus to vocal physiology, and much may still be learned from these new methods with respect to the fascinating signs of laryngeal paralysis. One new approach can be seen in Garde's chronaxic measurements on the recurrent nerve. Previously, chronaxie had been studied by Tarneaud, Murtagh and Campbell.

Cortical Representation of Laryngeal Movements

In 1884 Krause had detected the center for vocal cord adduction in the gyrus praefrontalis (praecrucialis) of the dog. Extirpation of this center produced aphonia of only short duration. The dogs so operated on did not bark spontaneously, while

all reflectory types of phonation remained undisturbed, obviously because the bulbar centers continued to function (Broeckaert). Subsequent research brought agreement with regard to the human cortical centers. The motor laryngeal area, also called area 6*b*, is situated in the lowest part of the cortex of the precentral gyrus, adjacent to the Sylvian fissure and extending to the area of Broca. Laryngeal sensation is represented in the corresponding region of the postcentral gyrus. However, Ónodi's hypothetical subcortical center has not been found. What he meant is probably identical with the bulbar centers (Lullies). Further observations during cortical stimulation were reported by Foerster. He correlated the area 6*b* with the masticatory region. All types of coordinated movements of the masticatory and articulatory organs could be elicited by its stimulation. Cushing reported regular phonation as a response to stimulation of the lowest part of the precentral gyrus.

Penfield and Rasmussen confirmed the areas of cortical representation by means of electric stimulation on human patients during brain operations with local anesthesia. They state that the representation of vocalization overlaps that for the movements of the lips, jaw, and the tongue. Vocalization was most frequently elicited from the motor area in the anterior central convolution, less often from the sensory area in the posterior central convolution. The patients so stimulated executed prolonged vowel cries for the entire duration of the stimulation. If necessary, they took another breath to continue the phonation. Stimulation of either side was equally effective, showing once more the bilateral representation of the cortical innervation of the larynx. In addition to this Rolandic area of vocalization, Penfield and Rasmussen also found a second region, which they called "superior frontal vocalization area." This lies in the frontal lobe within the longitudinal fissure. Its stimulation caused rhythmic or interrupted phonation and lip movements. When electrical stimulation was applied to these areas of vocalization after the patients had already begun to speak, speech was arrested. This is a negative stimulation response, comparable to the temporary paralysis of the extremities during their stimulation.

As a result of the bilateral representation of the cortical centers, and due to the partial decussation of the corticobulbar tracts, cortical lesions can produce laryngeal paralysis only if they are bilateral and symmetrical. Owing to the rarity of this prerequisite, laryngeal paralysis of cortical origin has not yet been definitely established (Foerster, Piquet). It is therefore very interesting that Lawson described a case of a left temporal lobe tumor which produced laryngeal anesthesia and paralysis of the cricothyroid muscles due to paralysis of both branches of the superior laryngeal nerve. There was no paralysis of the recurrent nerves, but rather adductor spasm, indicating their irritation. This observation tends to show that a unilateral cortical lesion may produce paralysis of the superior laryngeal nerves and bilateral spastic irritation of the adductors.

Cerebellar Centers

As for all voluntary movements, the cerebellum exerts a harmonizing influence on the tonus, balance, and fine coordination of the synergic as well as antagonistic innervation of the vocal organs. Thus, the cerebellum is responsible for the eurhythmia, eumetria, eutaxia, and diadochokinesis of all phonatory and articulatory movements.

Animal experiments and clinical observations produced some evidence for the localization of the cerebellar centers of laryngeal innervation. According to Katzenstein and Rothmann, a center for the innervation of the larynx and the masticatory muscles, and thus for phonation, exists in the lobulus centralis of the lobus anterior cerebelli (of the dog). Grabower contradicted these findings with other experiments on dogs. Further details were discussed by v. Skramlik.

Conversely, cerebellar disease may produce the atactic, asynergic, and dysmetric

disturbances of cerebellar dysarthria. This is characterized by exaggerated movements of respiration, overloud or interrupted phonation with nystactic ataxia of the vocal cords, iterative articulation, as well as disturbances of the rhythm and fluency of diction. The clinician describes these peculiarities of cerebellar dysarthia as scanning, chanting, or explosive (Luchsinger and Arnold).

Bulbar Centers

The laryngeal centers of the bulb are fairly well known since the studies by Semon and Horsley. They obtained in dogs opening of the glottis during stimulation of the ala cinerea, and vocal cord adduction during stimulation of the more caudally located calamus scriptorius. Further research (du Bois-Reymond, Katzenstein) attempted the precise localization of the various motor areas in the somatomotor nucleus of the vagus, which is the ventrally located nucleus ambiguus. In addition to this nucleus for the motor control of the striated muscles in the palate, pharynx, larynx, and esophagus, the vagus has a second motor nucleus, the dorsal visceral (parasympathetic) nucleus for the motor control of the heart, lungs, and intestines. The third nucleus lies laterally in the fasciculus solitarius. It has been called the sensory nucleus of the vagus. Von Skramlik believed that this nucleus received both the somatic and the visceral afferent fibers from all structures supplied by the vagus. According to the newest explanations by Kuntz, the fasciculus solitarius is reserved for the visceral afferent fibers, so that the somatic afferent fibers terminate in the nucleus of the trigeminospinal tract. This would be a fourth nucleus of the vagus system. In all probability the vagus does have four nuclei: (1) somatomotor for the striated pharyngeal and laryngeal musculature, (2) visceromotor for the smooth intestinal muscles, (3) somatoreceptive for the pharyngolaryngeal sensations, and (4) visceroreceptive for the intestinal sensations.

Analyzing the previous reports, Amersbach, v. Skramlik, and Stupka allotted the anterior third of the somatomotor nucleus ambiguus to the pharynx, esophagus, and to the cricothyroid muscle; the middle third to the palate, and the caudal third to the recurrent nerve. An arrangement of this type would agree with the autoptic and histologic findings in Luchsinger's case of isolated unilateral paralysis of the superior laryngeal nerve trunk and of the pharyngeal plexus, all of bulbar origin. Lasagna confirmed the cranial localization of the superior laryngeal nerve and the caudal representation of the inferior laryngeal nerve. N. Kruse quoted further corroborating cases and considered the site of the central representation of the recurrent nerve as definitely established, essentially as described above. It is interesting to note that the older authors occasionally described cases which agreed perfectly with our present concepts. Thus, Grabower quoted Wyscheslawtzewa's case (1909) of tabetic bilateral laryngeal paralysis; the vocal cords had been in paramedian position, and the degeneration of the nucleus ambiguus was most marked in its caudal portion. This example shows again that the older authors were usually correct in their observations but erred in their interpretations.

Experimenting on the chimpanzee, Furstenberg and Magielski verified the association of the anterior nuclear third with the cricothyroid muscle; the middle third was found to be the seat of abduction, while the caudal third appeared connected with adduction of the vocal cords. The agreement of these findings with those of Semon and Horsley is obvious. The opposite was claimed by Bouchet and Pialoux, who found in a case of abductor paralysis a lesion in the caudal third of the nucleus ambiguus. Albeit, the centers of abduction and adduction seem to be sufficiently far apart to account for the possibility of isolated bulbar paralysis of either of these opposite functions (Zlotnikov). This fact has also been used to explain the isolated lesions of either abduction or adduction in such bulbar lesions as syringomyelia, tabes, or multiple sclerosis (Broeckaert). While the concensus of opinions refers the superior laryngeal nerve to the cranial part of the nucleus ambiguus, its caudal parts remain problematic as far as the precise localization of the vocal cord movements are concerned.

Since the nucleus ambiguus is about 2 cm. long, it can be readily understood that a circumscribed lesion of its cranial portion will lead to an isolated paralysis of the palate, pharynx, and the cricothyroid muscle. This could be demonstrated by the cases of Fremel, Luchsinger, and those quoted by Kruse. On the other hand, a circumscribed lesion of the caudal part must be associated with an isolated paralysis of the recurrent nerve. This is understood to produce paramedian position of the affected cord. Again, there is good agreement with the old case of Wyscheslawtzewa and with the five cases of bulbar recurrent paralysis with paramedian position as described by Jepsen and Kristensen. If a third possibility were valid a bulbar lesion affecting both the superior and inferior laryngeal nerves would have to spread over the entire length of the nucleus ambiguus of the vagus. Theoretically, one could then expect the vocal cords to be in intermediate position.

Anatomy of the Vagus System

After leaving the oblongated medulla, the roots of the vagus nerve unite to form its intracranial trunk. Still within the jugular foramen the nerve enlarges to form the jugular ganglion. Several connections take place here with the facial, glossopharyngeal, accessory, hypoglossal, and the sympathetic nerves. After its passage through the jugular foramen the vagus forms the plexiform or nodosal ganglion which pertains to its afferent sensory and efferent parasympathetic fibers. Below this ganglion the pharyngeal branches of the vagus join the pharyngeal branches from the glossopharyngeal and sympathetic nerves to form the pharyngeal plexus. This plexus provides the principal motor and sensory innervation of the palate and pharynx. Only the M. tensor veli palatini is supplied by the trigeminus.

Slightly below the separation of the pharyngeal branches the superior laryngeal nerve leaves the vagus trunk. Embryologic considerations identify the superior laryngeal as the fourth branchial nerve. It divides into the internal branch, which supplies the laryngeal mucosa as far as the vocal cords, and the external branch for the motor supply of the cricothyroid muscle. In the middle of its cervical portion, the vagus trunk usually gives off the superior cardiac branch, an analogue of the fifth branchial nerve (Pernkopf). The inferior or recurrent laryngeal nerve separates from the vagus below the aorta on the left side, and below the subclavian artery on the right. This relationship of the recurrent laryngeal, being the sixth branchial nerve, to the sixth branchial artery on the left, and to the fourth branchial artery on the right, results from the embryonal origin of the aortic system. Thence the vagus trunk descends into the chest to distribute its motor, sensory, and secretory fibers to the visceral organs.

For phylogenetic reasons the accessory nerve follows a complicated course, which has caused much controversy. First, it consists of two distinct portions, namely, the cerebral (or bulbar) accessory and the spinal (or ascending) accessory nerve. The cranial or cerebrobulbar root has its origin in the lower caudal portion of the nucleus ambiguus of the vagus. The caudal or spinal roots of the accessory nerve, however, emerge from the descending accessory nucleus within the cervical medulla.

This arrangement shows again how the efferent and the afferent nuclei of the 9th, 10th, and 11th cranial nerves are coupled in series. The ventrally located somatomotor nucleus ambiguus of the vagus is a direct continuation of the more cranial motor nucleus of the glossopharyngeus nerve (Burger). Similarly, the nucleus ambiguus includes, more caudally, the nucleus of the bulbar accessory nerve. Hence its name. Precisely the same arrangement unites the visceral afferent nucleus of the intermedius nerve (facial) and its caudal neighbors, namely, the visceral afferent nuclei of the glossopharyngeus and vagus, all three per-

taining to the visceroreceptive center in the lateral tractus solitarius (Kaplan).

The cerebral accessory roots leave the bulb in the form of several filaments directly caudal to the roots of the vagus. These bulbar roots of the accessory nerve then unite to form its medial (or internal) branch, which merges with the vagus at or below the jugular ganglion. On the other hand, the spinal roots ascend through the occipital foramen and unite into the lateral (or external) branch of the accessory nerve. This lateral branch joins for a short distance with the medial branch of the accessory nerve. Below the jugular foramen, however, it separates again from the vagus to become the distinct trunk of the spinal accessory nerve proper. The further course to the sternocleidomastoideus and the trapezius muscle presents no problems. In addition to some cervical fibers from the segments C_2-C_4, these muscles derive their innervation mostly from the spinal accessory nerve by means of its external branch, and thus from the spinal or descending accessory nucleus. Strictly speaking, this innervation of muscles of the neck and shoulder by a spinal nerve, which first has to make a detour into and again out of the cranial cavity, represents another example of a "recurrent" nerve. Phylogenesis and embryology explain this paradoxon. Now it remains to clarify what becomes of the fibers of the cerebrobulbar roots after their internal branch disappeared within the vagus trunk.

The Vagoaccessory Problem

Since the historical development of the discussions on the vagoaccessory problem has recently been reviewed by Karcher and Buess, no space need be wasted with a recapitulation of this century-old controversy. These authors came to the following conclusions. On the basis of the nuclear arrangements, the course of the cerebrobulbar roots of the accessory nerve, the junction of its inner branch with the jugular ganglion of the vagus, and on the basis of the further peripheral route of the accessory fibers within the vagus, the medial or internal branch of the accessory nerve belongs entirely to the vagus. However, sections of the accessory nerve within the cranial cavity have not consistently been found to cause laryngeal paralysis. Therefore, these authors felt that certain discrepancies exist between morphologic and physiologic investigations of this problem.

Among those who opposed the view that the accessory nerve was connected with laryngeal innervation were Amersbach, Dorendorf, Gleitsmann, Grabower, Grossmann, Hajek, Lullies, Nagel, Negus, Ónodi, Rothmann, v. Skramlik. Other authors remained neutral like Eyriès, who recently considered this problem unimportant for vocal physiology. Sometimes the reason for disagreement seems to have been a semantic question. An example is seen in Matsumoto's correct description of the accessory roots, although he did not believe they connected with the larynx. Since every portion of the accessory nerve is known under at least two different names, it may well have been that the spinal accessory was meant by those (Grabower) who declined to see a connection of the accessory nerve with laryngeal innervation. Thus Truex and Kellner clearly speak of the spinal root of the accessory nerve when excluding it from the laryngeal nerves. On the other hand, King and Gregg specifically mention the spinal accessory as one of the sources of the laryngeal nerves. Semon used the term "pneumogastric spinal accessory" for the description of the physiologic unity of the two cranial nerves. In the anatomical chapter of the new encyclopedic "Otolaryngology" the internal branch of the accessory nerve is referred to as the "accessory portion of the eleventh nerve" (R. G. Williams). It certainly would have saved a great deal of unnecessary arguments had the revision of terminology been accepted as suggested by Ónodi.

The large majority, especially of the present investigators and teachers, advocates Claude Bernard's thesis of the accessory origin of laryngeal innervation: Broeckaert, de Beule, Della Cioppa, Dreyfuss, Elze, Font, Judson and Weaver, Kaplan, Kecht, King and Gregg, Kressner, Langendorff, Lederer, Musgrove, Pernkopf, A. Réthi, Rüedi, Semon and Horsley, Sieglbauer, Stupka, Tassi, Thomson and Negus, Titche. Minor disparities concern the question whether all motor laryngeal nerves are derived from the accessory nerve (Jackson

and Jackson, R. G. Williams) or whether the vagus does contribute some fibers (de Beule, King and Gregg) to the laryngeal muscles.

Such recent works as Scott-Brown's textbook of otolaryngology (Hamilton and Harrison) and Pernkopf's anatomy text assign all somatomotor fibers for the palate, pharynx, and larynx to the cerebral portion of the accessory. Della Cioppa, and Portmann described the vagus as a purely sensory and autonomous nerve, so that all motor fibers to the larynx would originate in the internal branch (from the cerebrobulbar root) of the accessory and merely borrow the vagus for their peripheral course to the larynx. Font believes that the accessory fibers are distributed principally to the pharyngeal plexus and to the superior laryngeal nerve. Kaplan concurs with this theory of the predominant vagal origin of the recurrent nerves.

A. Réthi's repeated animal experiments lend weight to the older concept of mixed and segmental laryngeal innervation by both the vagus and the accessory nerves. He believes in a double innervation of the laryngeal muscles with a variable pattern of branching and anastomosis. In his experiments the intracranial section of the accessory nerve was followed by degeneration of the majority of the recurrent nerve fibers. After the intracranial section of the vagus nerve, on the other hand, the motor fibers of the recurrent nerve were intact, while the motor fibers of the superior laryngeal nerve were found degenerated. Peripheral sections of the recurrent nerve and of the superior laryngeal nerve produced the same results as did the central interruptions. On the basis of these experiments, the superior laryngeal nerve appears to be a true vagal nerve, whereas the recurrent laryngeal comes from the bulbar accessory (de Beule, Gehuchten).

In conclusion of the foregoing it may be said that the cranial third of the nucleus ambiguus is the somatomotor center of the palate and pharynx. The motor impulses are transmitted by the rootlets of the glossopharyngeus and by the cranial roots of the vagus, and ultimately by the pharyngeal branches of the glossopharyngeus and vagus nerves.

Slightly more caudally in the nucleus ambiguus lies the center of the cricothyroid muscle. Its outgoing fibers again leave by way of the cranial vagal roots, and finally reach the superior laryngeal nerve. This is the fourth branchial nerve (Pernkopf) and thus corresponds to the more cranial portion of the somatomotor nucleus of the 9th, 10th, and 11th nerves.

The caudal parts of the nucleus ambiguus regulate the laryngeal movements of adduction and abduction. Consequently, the fibers arising from these caudal centers emerge from the bulb with the bulbar roots of the internal branch of the accessory nerve, which then joins the vagus at the jugular ganglion. Ultimately, these accessory fibers leave the vagus as the inferior laryngeal nerve. It seems logical that the recurrent laryngeal, which is the sixth branchial nerve, should pertain to the caudal portion of the vagoaccessory center.

In this way the accumulated evidence on the nuclear arrangement in the bulb, as well as on the physiologic relationship of laryngeal innervation, begins to present a more complete picture. It remains to be seen whether final agreement will favor this concept of a segmental motor innervation of the palate, pharynx, and larynx by the 9th, 10th, and 11th nerves. Although this arrangement appeals to logical considerations, such recent authors as Hamilton and Harrison, and Pernkopf still maintain that all, or most, motor fibers to these organs originate in the cerebral portion of the accessory.

Terminal Branching of the Recurrent Nerve

Whereas no one doubts the fact that the inferior laryngeal nerve carries the motor impulses to the intrinsic muscles of the larynx, the fine details of the terminal ramifications still remain controversial. This is explained in part by the variable types of branching. In most human cases some form of branching of the recurrent nerve trunk takes place before it enters the larynx, but the exact number of branches, the point of division, and the further route are variable. For this reason different observers may obtain different results of experimental sections of the recurrent laryngeal nerve or its branches. These differences would depend on the level of the interruption and on the individual pattern of branching.

The majority of authors agree with King and Gregg, Murtagh and Campbell, Semon, Winckler, etc., that the thick, heavily myelinated, ventral or anterolateral branch, or group of branches, supplies the adductor group of the intrinsic muscles, whereas the thin, less myelinated, dorsal or posteromedial branch, or group of branches, reaches the abducting posterior cricoarytenoid muscle. In addition, the dorsal branch is known to innervate the interarytenoid and the aryepiglottic muscle. It also joins the internal branch of the superior laryngeal nerve to form the sensory ansa Galeni. Only A. F. Williams took exception with this concept and believed in a motor and sensory separation of these fibers. According to his findings, the ventral branch would innervate all intrinsic muscles motorically, while the dorsal branch conveys the sensory supply of the lower larynx, and joins the superior laryngeal nerve by way of Galen's ansa. Roberto merely enumerates five end-branches, four motor, and one to the ansa Galeni. Following the clear presentation by Pressman and Kelemen one can summarize the definite and the controversial problems of terminal ramifications in the following manner:

1. The recurrent laryngeal nerve supplies unilaterally all intrinsic muscles of the larynx. The external branch of the superior laryngeal nerve supplies the external cricothyroid muscle (de Beule).

2. It is agreed that the interarytenoid muscles are innervated bilaterally by both recurrent laryngeal nerves (de Beule, Grabower).

3. Many times the internal branch of the superior laryngeal nerve has been assigned a part in the motor innervation of the adductors (Graceva, Klestadt, Moran, A. Réthi, Rubaltelli), and particularly of the interarytenoid muscles (Freedman, Imperatori, Kressner, Lore, Negus, New and Childrey). Recent views held that these fibers are of a purely sensory nature (Hofer and Jeschek), inasmuch as they have been found to perforate the muscles (Grabower) in order to reach the mucosa of the aryepiglottic folds (Lemere, Winckler). However, Berendes, Clerf and Baltzell, Todd, and P. H. Vogel contradicted this concept anew by demonstrating motor end-plates in the terminal ramifications of the superior laryngeal nerve of man. Thus, they believe in a double motor innervation of the interarytenoid muscle in man. Animal experiments failed to corroborate these findings (Lemere, Murtagh and Campbell). In further human experiments with electric stimulation of the superior laryngeal nerve a motor response of the interarytenoid muscle could not be elicited (Meurman, A. F. Williams). These considerations led Pressman and Kelemen to believe that the interarytenoid muscles do not receive motor fibers from the superior laryngeal nerves.

Present Interpretations of Semon's Rule

It is now widely accepted that the section of all fibers of one recurrent laryngeal nerve is followed by the median or paramedian position of the vocal cord on that side. This is distinctly different from the intermediate or semiabducted position because the cricothyroid muscle retains its innervation by the intact external branch of the superior laryngeal nerve and produces an adductor effect and a sufficient amount of tension of the vocal cord.

Intermediate position with bowing, flaccidity, and shortening of the paralyzed cord, and a tilting forward of the same arytenoid cartilage, occurs only after the additional interruption of the superior laryngeal nerve trunk or its external branch. This is called "essentially complete" and combined paralysis of one side of the larynx. Because of the bilateral innervation of the interarytenoid muscles no absolutely complete paralysis can be obtained from unilateral nerve interruption.

Innumerable experiments have verified Grossmann's claim that a median position can be changed into the intermediate position by the additional interruption (by anesthesia or section) of the external branch

of the superior laryngeal nerve or of the cricothyroid muscle directly (Arnold, Freedman, Grabower, Killian, Lemere). Contradicting evidence was due to the fact that the cricoarytenoid joint becomes rigid (but not necessarily ankylosed) after a certain duration of the paralysis. Messerklinger and Propst recently investigated this problem. When, therefore, an articular fixation complicates the median position, the interruption of function of the cricothyroid muscle will no longer produce a relaxation of the cord in the intermediate position. As can be noted, the term "fixation" has been reserved for the state of limited motion in the cricoarytenoid joint. This well-known distinction between the articular fixation of the arytenoid and the paralytic loss of its muscular movement has also been stressed by Grotting and Pemberton.

Obviously, the original observations of the earliest experimenters, such as Legallois (1812) and Longet (1841), and the explanation by Wagner and Grossmann appeared to be correct, while Semon's interpretation of the median position as a partial paralysis of the recurrent laryngeal nerve had to be modified in favor of that of his opponents. On the other hand, his claim of the distinct proclivity of the abductor fibers to become affected earlier than the adductor fibers had been confirmed by clinical experience. It was this twofold problem of Semon's law, namely, his (erroneous) interpretation of the midline position as a partial recurrent paralysis and the (correct) observation of the greater susceptibility of the abductor fibers, which caused so much confusion and controversy.

Many theories had been proposed to explain Semon's rule. While each one was based on correct experimental evidence, few explanations sounded as convincing as the one offered by Negus. He pointed to the fact that primitive functions are more resistant to damage than are the phylogenetically younger functions. The function of the adductor muscles corresponds to the primary sphincter action of the larynx of low vertebrates which became necessary for the protection of the pulmonary airways. In contradistinction, the abductor function represents a more recent perfection of the inspiratory relaxation of this protective sphincter mechanism. Therefore, the abductor function would be more readily damaged, so that the persistent position of adduction of the cords in laryngeal paralysis could be interpreted on a phylogenetic basis.

Furthermore, the posticus muscle is the lone antagonist of all other intrinsic muscles of adduction. In addition, its continuous activity is concerned with the constant function of respiration, whereas the intermittent contraction of the adductors occurs only during the short periods of expectoration, deglutition, and phonation. Besides these gross differences of alternate antagonism, there is the permanent antagonism of the tonic respiratory oscillations of the cords. As is well known since the studies of de Beule, Murtagh and Campbell, A. Réthi, Stupka, etc., the abductors provide an additional widening of the open glottis during inspiration; they are the inspiratory muscles. During expiration the adductors, including the external cricothyroid, produce a slight narrowing of the open glottis; they are the expiratory muscles. The reversal of this fundamental principle is known as perverse action of the cords, which is a sign of psychogenic or neurologic disturbances: functional inspiratory stridor, inspiratory phonation, etc. In view of these facts it is really amazing that no one ever complained of laryngeal fatigue from constant breathing. Yet, even the best singers and speakers cannot avoid a certain amount of vocal and laryngeal fatigue after, say, an hour's vocal exertion. Expressed in phylogenetic terms, the most recent function of phonation is subject to fatigue, whereas the second oldest function of respiratory abduction is not.

All these fundamental differences between the opposite functions of adduction and abduction tend to explain why the single pair of posticus muscles should become affected more readily than their antagonistic group of the adductors. Finally, there may be a parallelism with other paralytic conditions in which nature attempts survival at a lower level of efficiency. In hemiplegia, for instance, the spastic paralysis immobilizes the arm in flexed position, while the leg is held rigid in spastic extension. Obviously, this situation is more advantageous for the invalid than any other combination of paralytic rigidity would be. From this aspect it seems plausible that nature prefers a limitation of respiration by limited abduction rather than to lose the deglutitory protection of the airways as a result of impaired vocal cord adduction.

In evaluating Semon's rule one must bear in mind two facts: one, that he spoke of slowly progressing lesions, and not of experimental sections; two, that isolated abductor paralysis is not identical with the midline position of the cords (Grossmann)! A great deal of confusion had been caused by this faulty assumption, especially when certain cases could not be forced into that system. Fabry questioned a case of L. Lack: "complete bilateral abductor paralysis" with median position since suicidal transsection of the trachea; how could a bilateral interruption of both recurrent nerves (below their ramification) damage primarily the medioposterior branches to the abducting posticus muscles, leaving the anterolateral branches to the adductors intact? Now we seem to know better; this must have been a case of complete paralysis of both recurrent nerves.

Every type of two antagonistic functions shows two opposite extremes, and an intermediary position of neutral relaxation or balance. In the case of the vocal cords one recognizes the midline position of complete adduction for phonation, and the lateral position of complete abduction for deep inspiration. Let us first analyze the movement of abduction. When the adductor muscles relax, the cords follow their arytenoid cartilages, which slide down along the inclined articular surfaces of the cricoid. A neutral position of relaxation is then reached in the intermediary line or position. From this point the abducting posterior cricoarytenoid muscles come into play and pull the cords lateralward until the lateral position of full abduction is reached. Conversely, the relaxation of the posticus muscles allows the arytenoid cartilages to rotate back to the intermediary position. From there the cords are then brought to the midline by the active contraction of the adductors. The constant tonus of all intrinsic muscles maintains the proper balance in the interplay of this complicated coordination, and slight fluctuations of this tonus can be seen in the reflectory oscillations of the cords during quiet respiration.

Pure abductor paralysis disturbs neither the active adduction of the affected cord from the intermediate line to the midline position nor the passive return of the cord from the phonatory position to the intermediary line of relaxation. What is lost in pure abductor paralysis is the full active abduction of the cord from the intermediary line to the lateral position of deep inspiration (Broeckaert, Dorendorf, Hajek, Klemperer, Stupka, Weleminsky). This has often been seen as the first paralytic sign of a slowly progressive lesion of the central or peripheral pathways of the inferior laryngeal nerve (Fabry, Freudenthal, Michaelis, Russi). Conversely, the same is true for the last stage of a subsiding recurrent paralysis of traumatic or toxic origin (Roy). Adduction having recovered first, abduction remains sluggish for a time. When abductor paralysis is seen in this light, there is no need to doubt the validity of Semon's rule. As

far as it concerns the greater vulnerability of the abductor function, it represents a clinical observation and directs our attention to the possibility of an incipient and possibly progressive paralytic lesion.

Conclusions

Although the problems connected with laryngeal innervation are very numerous, and although the large number of studies on this subject accounts for the great diversion of opinions, sufficient agreement on the main issues has already been reached.

Contrary to investigations on the highly developed hearing organs of animals, the value of animal experiments for human phonation appears limited. The usual laboratory animals differ significantly from man in regard to laryngeal anatomy and physiology. Only the primates show a closer resemblance to human anatomy. However, from the physiologic viewpoint, no animal is adequate for a complete demonstration of human phonation, simply because the vocal performances of animals do not remotely approach the high degree of differentiation of the human voice. A better understanding of human laryngeal innervation was gained after suitable experiments were undertaken during laryngologic operations.

This reservation is particularly true with respect to the cerebral representation of laryngeal functions in man. Although some basic orientation could be achieved by operating on the considerably more primitive cortex of animals, the only significant findings were made during human brain operations. Further neurosurgical investigations of the various aspects of human phonation appear most promising of new results.

While it is established that the vagus is ruled by four bulbar nuclear areas (somatomotor, visceromotor, somatic afferent, and visceral afferent), the precise localization of the various somatomotor actions of the vagus is not completely understood. It seems logical that the movements of the palate, pharynx, and of the cricothyroid muscle should be represented in the cranial portion of the somatomotor nucleus ambiguus of the 9th, 10th, and 11th nerves. Embryologic, anatomic, and physiologic considerations, as well as pathological findings, make it appear equally logical that the caudal portion of the nucleus ambiguus should be the center for the intrinsic laryngeal movements.

The internal branch of the accessory nerve is an integral part of the motor vagus. However, the opinions still differ with respect to the precise course of the accessory fibers to the muscles of the pharynx and larynx. Considering the arrangement of the bulbar nuclei, it would appear logical that those experimental findings are correct which describe the superior laryngeal nerve as a true vagal branch. For the same reasons the inferior laryngeal nerve appears to be derived from the accessory nerve. It remains to be seen whether final agreement will favor this concept of a segmental motor innervation of the palate, pharynx, and larynx by the 9th, 10th, and 11th nerves.

The terminal branching of the recurrent nerve is very variable. These individual variations account in part for the many contradictions in previous studies on laryngeal innervation. Otherwise, they seem to be responsible for the variety of clinical findings in laryngeal paralysis. In particular, the problem of a motor supply of certain intrinsic muscles, especially of the interarytenoids, by the internal branch of the superior laryngeal nerve is not definitely solved. However, the majority of authors reject this assumption.

Complete paralysis of the recurrent nerve is now understood to produce median or paramedian position of the paralyzed cord. Intermediate position of

a bowing and atrophic cord is interpreted by most authors as a sign of complete and combined paralysis of both the superior and inferior laryngeal nerves.

Pure abductor paralysis limits only the full abduction of the affected cord from the intermediary line of relaxation to the lateral position of deep inspiration. This can be seen either as a first sign of an incipient lesion of the vagus system or as the last stage of a subsiding paralysis.

After Semon's law had been disapproved by the majority of authors, it is now understood that Semon's observation of the greater vulnerability of the abductor fibers represents a clinical sign. As such it attracts the attention to the possibility of an incipient and possibly progressive paralytic lesion.

Summary

An attempt is made to define the major problems of laryngeal paralysis by analyzing the representative bibliography from the following viewpoints: value and limitation of animal experiments; chronologic changes in the concepts of laryngeal paralysis; discussion of the central pathways and the peripheral course of the laryngeal nerves; distribution of vagal and accessory fibers in the laryngeal nerves; variability of the terminal ramification of the recurrent nerves, and the present interpretation of the position of the paralyzed vocal cords.

The majority of authors favor Grossmann's original concept: Median position means complete recurrent paralysis, while intermediary position is interpreted as a combined paralysis of both laryngeal nerves.

Semon's rule of the greater vulnerability of the abductor fibers represents a clinical observation which signifies either an incipient sign of a progressive lesion or a terminal sign of a subsiding paralysis.

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The Frontonasal Angle in Cosmetic Rhinoplasty

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The creation of an esthetic profile in cosmetic rhinoplasty not infrequently demands the formation of an adequate frontonasal angle. The contour which comprises the end-result of simple rhinoplasty in its relation to the frontonasal angle historically permits some distinctly varying preferences based on cultural background. The desired contour will, of course, vary with each person. Variations also exist which are based on the particular preferences of different cultural groups. For example, in many Near Eastern countries, profiles with little or no frontonasal angles are admired, and postoperative results, although highly satisfactory to them, do not entirely fulfill our esthetic standards. Ideal profiles, again, have found their variations in different eras. A "Grecian profile" or a "Roman nose" may have been desirable at one time. It is not necessarily the model now for the esthetic profile (Fig. 1).

Holden states that Dr. Jaques Joseph did extensive research in order to obtain good insight into this subject. He examined a great number of busts and portraits from the Greek and Egyptian eras. The variations in the frontonasal angles which he encountered apparently were rather significant and were decisive influences in his work the remainder of his life.

In the majority of rhinoplasties performed today the problem of creating a desirable frontonasal angle is not overwhelming. Where the angle is weak or absent preoperatively, the problem takes on greater magnitude and requires more energetic procedures. Rhinoplasty, as usually described, consists in the removal of the nasal "hump" by bone-biting forceps, bayonet saws, and a variety of osteotomes. Most of these techniques describe the complete removal of the nasal hump, but always superficial to the level of the nasion (Fig. 2). This procedure, fortunately, is satisfactory for the majority of noses submitted to surgical correction.

However, about one-third of the cases have a decreased or absent frontonasal angle, requiring a modification in surgical management. For several years, we have practiced the removal of nasal humps so

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Fig. 1.—Examples of beauty demonstrating absence of frontonasal angle: (Left) Queen Nefertiti, Egyptian, circa 1350 B. C. (Right) Venus de Medici, Greco-Roman, circa 200 B. C. (Courtesy of Metropolitan Museum of Art.)

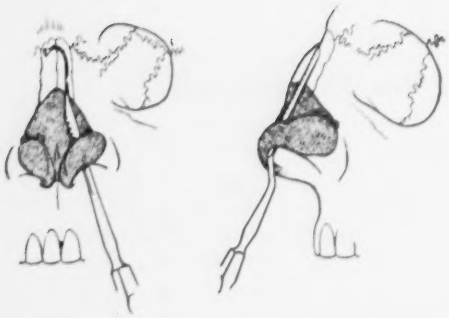


Fig. 2.—Conventional method of removing nasal "hump" superficial to nasion.

Fig. 3.—The osteotome is used to cut below the surface of the glabella. When properly manipulated, as indicated by arrows, the nasal bones will be fractured or disarticulated so as to include the glabella.

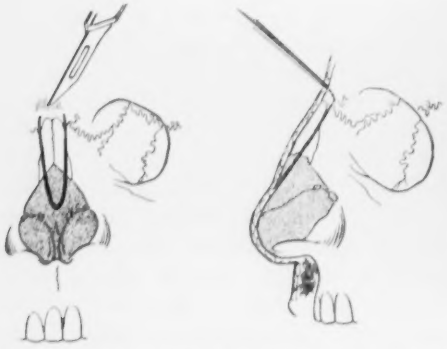


Fig. 4.—An incision with insertion of a 2 mm. osteotome to cut the bone at a right angle to the original cut, so as to include a portion of the glabella with the specimen.

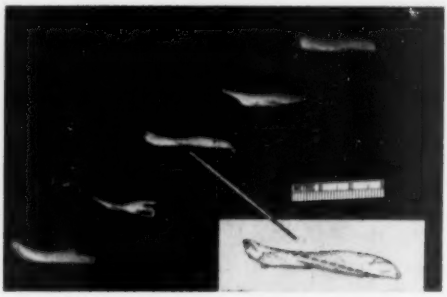
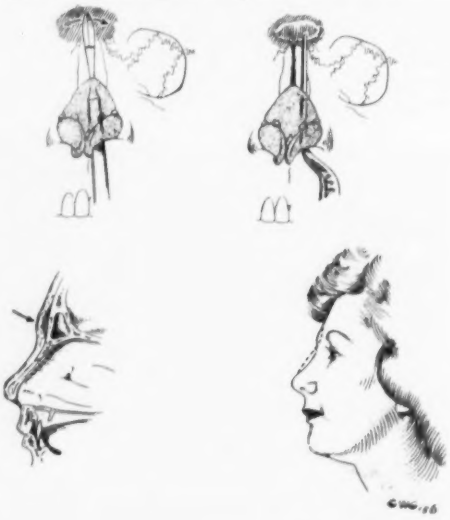


Fig. 5.—Representative nasal "humps," including the glabellar portion obtained by methods described.

Fig. 6.—Creation of a pocket in the glabella region with insertion of a graft.



as to include the frontonasal angle in the specimen.

Technique

Several methods have proved satisfactory in our hands. Saws may be used to remove the nasal "hump," cutting below the surface of the glabella. This must then be followed by an osteotome which is carefully manipulated so as to cause fracture or disarticulation of the nasal bones with the attached glabella (Fig. 3). The osteotome alone may be used. It can be guided to a level again beneath the glabellar surface and fracture accomplished as above. In certain cases where there is complete absence of the glabellar angle and removal of a larger amount of bone is required, fracture by

Fig. 7.—Postoperative results utilizing the methods described. Cases A and B represent insertion of nasal bone in the forehead area.

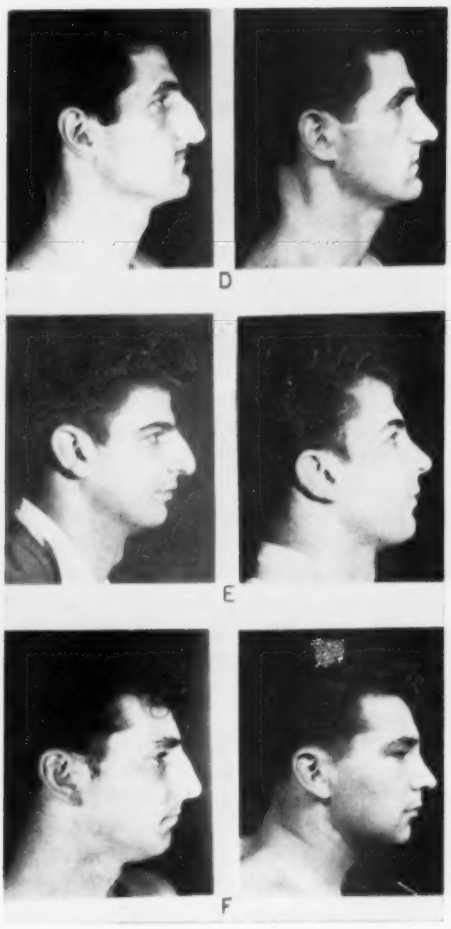


Fig. 8.—Postoperative results from the use of the methods described.

manipulation may not be possible. Under these circumstances, a 2 mm. incision in the glabella area is recommended, through which a very fine 2 mm. osteotome is inserted to complete the fracture (Fig. 4). The minute incision in this area requires no suture and is seldom discernible postoperatively. Figure 5 represents examples of nasal "humps" removed by the above methods.

Further increase in the relative frontonasal angle may be desirable in some extreme defects. This may be accomplished by building up the forehead in the glabellar region so as to further increase the relative nose-forehead proportions obtained by the above-mentioned methods (Fig. 6). This continuation of the nasal skeletonization must include the glabellar region. A pocket is thus created, which is then filled either with preserved costal cartilage from the bank or with the patient's own nasal bone. Cases A and B in Figure 7 represent insertion of patient's nasal bones in the forehead area.

Failure to attempt creation of a satisfactory frontonasal angle will result in dissatisfaction both to the surgeon and to the patient.

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Dorsal Sympathectomy in Labyrinthine Disease

A Review

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In 1954 a review of the subject of dorsal sympathectomy in labyrinthine disease was promised by the late Leighton F. Johnson, M. D.,[1] but because of his untimely death this was not possible for him. The cases which he treated have been followed, and to their number others, who were treated in like manner, have been added. A review of the results is now possible.

The champion of this form of treatment of Ménière's disease and allied conditions was Passe; the final summary of Passe's work,[2] which was published posthumously, showed that he obtained an approximately 60% favorable response to this type of treatment.

In 1954 Lewis[3] reported his results in 17 cases of Ménière's disease subject to incapacitating attacks of vertigo. In 7 of the 17 cases he obtained satisfactory control of the attacks. The remaining 10 cases were either failures or received insufficient benefit from the operation. Five of the seventeen cases were found to have abolition of, or satisfactory reduction in, the preoperative tinnitus. The remaining 12 either were unaffected by the operation or had a relapse following temporary improvement. Lewis was not convinced that the operation had any significant effect on the hearing acuity. Any apparent improvement, as recorded by pure-tone audiometry, he felt, may have been explicable on the grounds of the typical spontaneous variations in the threshold of hearing that is known to occur in Ménière's disease. He felt that there was insufficient evidence to state that a deterioration of hearing can be prevented by dorsal sympathectomy. In his experience the value of preoperative stellate ganglion block with the use of procaine was doubtful. He found it impossible to predict in which cases a satisfactory result might be anticipated.

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Experimental Evidence

The rationale of this method of treatment of Ménière's disease and related conditions is not well founded.

It has been thought by many workers in this field that imbalance of the autonomic nervous system, and in particular a preponderance of a sympathetic component, was responsible for changes in the labyrinth found in Ménière's disease. It is known that sympathetic nerve fibers reach the internal ear along with the internal acoustic artery, and Smith[4] reported the finding of numbers of sympathetic fibers around the primary and secondary branches of the cochlear artery in the modiolus. The distribution of efferent fibers, probably entirely parasympathetic, in the internal ear has been described by Fernandez[5] and Rambo;[6] some of the fibers have been seen to reach the internal hair cells of the organ of Corti.

Although the cause of Ménière's disease is not known, it must be apparent to all who take care of patients afflicted with this troublesome condition, that they all have at least one finding in common. Patients with Ménière's disease are of the nervous, apprehensive, tense type of personality; they are overconscientious, worry too much, and work too hard. The relationship between

psychological factors and the occurrence of episodes of labyrinthine disease is unquestionable. Frequently patients will volunteer the information without being asked that the onset of their attacks of vertigo, or the beginning of a new series of attacks, dated back to a particular mental or emotional disturbance. These occurrences have been too frequently documented to be mere coincidence.

It should be pointed out, however, that although these patients show evidence of emotional instability, they do not have mental disease. Earlier in this present investigation a small number of cases were investigated by the department of psychiatry at Boston University School of Medicine, and it was definitely felt that these patients were not mentally ill and would not benefit from psychiatric care. The most that could be said of their mental condition was that they tended to have evidence of a mild anxiety state.

Undoubtedly psychic stimuli reach the hypothalamus, and from this center there is an available pathway over the sympathetic and parasympathetic systems to the vasculature of the internal ear. Whether these pathways are of significance in diseased states, is hypothetical. There are several fragments of evidence which point to the fact that certain disease processes of the internal ear may be affected by intervention in the sympathetic nervous system.

Seymour and Tappin[7] have shown on a series of experiments on cats that stimulation of the cervical sympathetic nerve trunk produced a diminution in the size of the cochlear microphonic potentials. Histologic examination of the stimulated cochlea showed concavity of Reissner's membrane, suggesting that there had been a diminution of the secretion of endolymph. In another series of experiments they were able to show, in the blood vessels of the living stria vascularis, that the vessels contracted on sympathetic stimulation; the contraction was seen to be not so well marked as in the extracranial vessels.

Fowler[8] has described an interesting phenomenon found in peripheral capillaries known as sludging of the blood, which can be induced by a variety of psychic stimuli. These changes have been examined in detail in the conjunctival mucosa, and these same changes have been described and seen shortly before and after attacks of Ménière's disease. It is not possible to state, of course, that such a change does actually occur in the minute vessels of the inner ear, but certainly there appears to be no reason why such a change should not occur.

On the other hand, Rambo and his associates[6] failed to produce any change in the internal ears of monkeys after completely interrupting either the parasympathetic or the sympathetic supply. They also failed to demonstrate any dilatation of the blood vessels of the perilymphatic space after complete sympathetic denervation in a monkey under general anesthesia.

In 1953 I undertook a series of experiments in collaboration with Professor Lutz in the department of biology at Boston University. The project was for the purpose of determining, if possible, any demonstrable influence that the sympathetic system might exert over the labyrinthine vessels. As the experimental animals, dogs weighing 10 to 15 kg. were selected because they were readily available. Animals of this size possess the disadvantage that the labyrinth is small, being approximately half the size of the human labyrinth. Intraperitoneal barbiturates were used as the anesthetic agent.

It is a simple matter to expose the mastoid process of the dog's temporal bone, and careful removal of the cortex and underlying cancellous bone with a drill will expose the most superficial semicircular canal of the labyrinth.

After a little practice I found that I could create a smooth, well-defined fenestra on the convexity of the semicircular canal, using a dissecting microscope with 30 × magnification, without damaging the membranous labyrinth (Figure). I used a con-

Fenestra in labyrinth of a dog; reduced 2/5 from 30 × magnification. Note the large marginal vessels, through which the blood flow was constant. The flow through the smaller communicating vessels was intermittent and often reversed its direction.

tinuous irrigation of isotonic saline at a temperature which was within 3 degrees (centigrade) of the animal's body temperature. In this way I could see one or two relatively large caliber vessels crossing over the surface of the membranous labyrinth, through which blood was flowing at all times. As well as these two larger vessels there were many much smaller vessels, through which the blood flow was intermittent. Not only was the flow intermittent, but at times the direction of the flow would be reversed. There seemed to be no constant pattern in the distribution of these vessels over the dog's labyrinth from one dog to another. While acquiring the necessary dexterity to make these small fenestrae, which measured approximately 1.5×0.5 mm., it soon became apparent to me that many factors have an influence upon the flow of blood through these minute vessels in the exposed labyrinth.

The temperature of the irrigation fluid was of extreme importance. When the temperature fell below 30 C the vessels would go into a spasm, so that only the major vessels were seen to contain blood. If the temperature of the irrigating fluid was returned to approximately body temperature, the small vessels would reopen. However, if the vessels were cooled below 20 C, they would go into spasm and the process would be irreversible. Similarly, increasing the temperature of the irrigating fluid above that of the body would cause the vessels to dilate. On lowering the temperature again the vessels would often constrict to their normal size.

The tonicity of the irrigating fluid was of great importance, because if hypotonic fluid were used instead of isotonic saline, even though the temperature was close to body temperature, the vessels would go into spasm and could not be reopened. A similar effect was found by using hypertonic fluid. Should the flow of the irrigating fluid be inadequate and the fenestra was allowed to become uncovered, the heating effect from the source of illumination of the microscope would be sufficient to first dilate the vessels and then put them into permanent spasm, so that they could not be reopened.

These observations immediately suggested the various difficulties which would be present in trying to demonstrate any control that the sympathetic system might exert over these minute vessels of the inner ear.

Exposure of the vagosympathetic trunk is simple, and at the lower end of this trunk where the sympathetic nerves separate from the vagus, the stellate ganglion is found lying at the posterior end of the first intercostal space. Having exposed the vessels of the labyrinth, I would expose the sympathetic trunk and stellate ganglion, after which I would first stimulate the ganglion with a faradic current and watch for any visible change in the caliber of the vessels or the rate of blood flow. On six occasions, where I had a satisfactory exposure of the

membranous labyrinth without injury, I was unable to demonstrate any changes in these vessels during stimulation of the stellate ganglion or the vagosympathetic trunk.

Blocking the stellate, using at first procaine and then surgical section, was similarly found on six occasions to have no visible effect on the vessels of the membranous labyrinth.

Following these findings it was decided to give a trial to the effect of intravascular injections of vasodilators and vasoconstrictors in order to see if the vessels could be altered in this way. Epinephrine, 0.5 cc. of a 1:20,000 solution, was injected into the vertebral artery and then into the carotid artery, without any visible change appearing in the vessels of the membranous labyrinth. During injection of the carotid system blanching of the conjunctiva and buccal mucosa was noted consistently. After this a similar experiment was conducted using 10 mg. of sodium nitrite in 1 cc. of isotonic saline, and this fluid was injected into, first, the vertebral artery and then the carotid system, without visible effect upon the labyrinthine vessels. Marked extracranial vasodilatation was brought about, as evidenced by brisk bleeding from the edges of the soft tissues of the incisions.

Following the lack of response to intravascular vasoconstrictors or vasodilators it became apparent that further experimentation and intervention in the sympathetic system would not be rewarding. In retrospect this finding should not be very surprising because Smithwick [9] has found that it is almost impossible to obtain any useful results in experiments designed to determine function of the sympathetic system in any part of the experimental animal when general anesthesia is used. Wilkins [10] has commented upon the vasodepressor effect of barbiturates and believes it must be on the basis of vasodilatation. This then would appear to be the basis of the problem, namely, that no conclusive results can be obtained one way or the other, unless an acute experiment can be designed in which anesthesia is not necessary.

At this point it would be relevant for me to recall the finding of Passe [2] when he performed a stellate ganglion block using procaine during a fenestration operation under local anesthesia. At that time he recorded that he distinctly saw gross and obvious dilatation of the vessels of the membranous labyrinth. Regrettably, no one has had opportunity to confirm or refute this finding.

The Problem

Before presenting the results of our experience with the treatment of Ménière's disease and allied conditions by intervention in the sympathetic system, I should like to emphasize several factors that are encountered which present a very real problem in trying to evaluate the effect of any treatment in these conditions.

In Ménière's disease in particular these patients are notoriously unreliable in the way that they record their experiences and are extremely poor observers of their own difficulties and sensations. This situation is further complicated by the fact that the disease is characterized by the occurrence of spontaneous remissions of undetermined length. These factors then make it extremely difficult to present an unbiased view of the efficacy of any one particular treatment.

In examining the records of these patients one is immediately struck by the astonishing discrepancies in the observations made by two or three physicians who examined a patient on the same day; I am convinced that it is nigh impossible to get a consistent story from these patients when one is trying conscientiously to form an objective opinion on their progress.

Method of Selection of Cases

The cases chosen for this study were selected on the basis of poor or no response to conventional medical treatment so that all would have been potential candidates for

destructive labyrinthotomy, although in some cases the hearing was still so good as to preclude this treatment.

In all cases the effect of a procaine block of the stellate ganglion was tried to see if any change could be effected in the tinnitus or hearing. The usual anterolateral approach to the ganglion was used, and about 8 cc. of 1% procaine was usually required to produce a temporary Horner's syndrome. An attempt was made to make this a test of prognostic significance.

If it appeared from the accumulated evidence that intervention in the sympathetic system had a reasonable chance of being helpful, dorsal sympathectomy was advised and performed. The operation used was the standard preganglionic section and excision of the dorsal ganglia 2, 3, and 4. The stellate was not removed because it is thought that few, if any, fibers from the first dorsal go to the head, except those to the eye. None of the patients had a Horner's syndrome after the operation.

All the patients had preoperative vascular studies carried out to provide a comparison for postoperative studies so that an estimate could be made of the completeness of the denervation.

Cases Treated by Dorsal Sympathectomy

In presenting the results of this treatment of labyrinthine disease I have tried to give an objective view of the efficacy of the procedure. The whole concept has been extremely controversial for several years, and we set out to find out once and for all whether or not the treatment was worth while. Three years ago when we started this investigation we were quite naturally very enthusiastic about the prospects, but we cautioned that experience had been limited and that time alone would tell the final outcome. In all the cases two or more years have passed since the operation, so that a judgment can be passed on the results. I have been purposely very exacting in my criteria of a successful outcome—some would say too exacting. However, I will present the findings in what I believe to be their true light.

TABLE 1.—*Cases Treated by Dorsal Sympathectomy*

	Ménière's Disease	Tinnitus
Number of cases treated	9	4
Initial successes	5	2
Initial failures	4	2
Permanent successes	1	2
Late failures	8	2

The average onset of relapse of Ménière's disease was eight months.

There were nine cases (Table 1) of Ménière's disease which had a dorsal sympathectomy; the indication for operation in all was intractable vertigo which could not be controlled by medical therapy. In addition, one case had an acute severe deafness of approximately 70 db. which had been present for only two weeks before operation.

There were four cases in which the indication for operation was severe tinnitus without vertigo. In one of these cases the accompanying nerve deafness did not exhibit recruitment.

Effect of Sympathectomy on Vertigo

Of the nine cases of Mèniére's disease, only five were initially successful; they had no attacks of vertigo at all for three or more months. Of the four remaining cases, they all continued to have attacks of vertigo immediately following operation and were complete failures. One of these latter cases had the other side operated upon two years later and was improved, although barbiturates were still required to keep her under control.

Late review of these cases shows that of the five cases initially successful only one has remained free of attacks and requires no medication; the other four continue to have minor attacks or continue to need medication. The bilateral case has no further attacks as long as barbiturates are maintained.

Thus there are six cases out of the nine who are pleased with the result of the operation in that they are better than before operation. However, I consider that only the one who has had no attacks and requires no

medication can be designated a complete success.

Effect of Sympathectomy on Tinnitus

The four cases of tinnitus were more responsive to dorsal sympathectomy; two cases have had no further tinnitus and two have continued as before operation. It is of interest to note that both the cases who responded to operation had had severe vascular headaches before operation and that these also disappeared after operation. One of the failures was a case of Raynaud's disease who had no recruitment and did not respond to procaine block of the stellate ganglion; it was no great surprise that the tinnitus continued.

Effect of Sympathectomy on Hearing

One feature of this type of treatment which made it attractive was that not only did it not destroy the hearing but there appeared to be a possibility that the hearing would in fact be improved. Because the disease is characterized by marked spontaneous fluctuations in the hearing level, any change in hearing must be great before it can be attributed to the treatment.

In one case with a 75 db. loss in the conversational frequencies the hearing returned to normal 10 days postoperatively. The deafness had been present for a very short time—about two weeks—and this may be the key to this astonishing and gratifying response. The patient has maintained normal hearing for three years.

In another case with a long-standing loss, 60 db., the hearing improved to the 20 db. level postoperatively; this improvement was maintained for 18 months, when vertigo recurred and the hearing fell to the 45 db. level.

Other cases showed improvement, but it was not sufficiently great to be attributed directly to the sympathectomy. There was one case which is of great importance because the hearing did not improve after operation and 18 months postoperatively the hearing deteriorated to the 45 db. level in spite of operation. It is unfortunate that this occurred in the one case which had a poor sympathetic denervation following surgery, as determined later, and who was an immediate failure clinically.

Onset of Relapse

Perhaps the greatest difficulty in treating Mèniére's disease is to effect lasting relief; many of the popular remedies will produce a remission in the disease for a time, especially if the physician happens to have great enthusiasm for the particular treatment. Oftener than not the long-term results are disappointing.

There were four cases of Mèniére's disease which did very well initially in that they had no attacks of vertigo; after the passage of several months, however, symptoms began to recur. The recurrent symptoms were less than the preoperative ones, and the patients were convinced that the operation had helped them. The onset of relapse varied from 3 to 18 months, the average time of onset being 8 months.

Outcome in Relation to Degree of Sympathetic Denervation

Whether relapse was due to return of sympathetic function or to the wearing-off of the full psychologic impact of the operation is not known. An attempt was made to solve the mystery by carrying out postoperative studies on the amount of residual or returned sympathetic function in the upper limb. Tests of sympathetic function are dependent on three main reactions: the lowering of surface temperature by vasoconstriction when the body is in a cold environment; lowering of pulse volume by vasoconstriction in response to cold as evidenced by plethysmography, and the vasoconstriction in response to noxious stimuli, such as pain, noise, and contact with ice. The state of the upper limb sympathetics is only presumptive evidence of the state of the cranial sympathetics and, in particular, those of the internal ear.

Seven cases could be persuaded to have these studies (Table 2). Three were found to have no residual sympathetic function of the upper limb (indicating satisfactory denervation), three had slight residual function, and one had excellent function in both arms after bilateral operation. The explanation of this residual function is controversial and three factors are offered: inadequate operation due to unknown nerve pathways; regeneration of nerve fibers, and occurrence of sensitization of denervated organs to circulating substances, such as epinephrine.

Three cases had no demonstrable sympathetic function in the arm; one was a case of tinnitus who had remained free of tinnitus, one a case of Mèniére's disease who was initially relieved of attacks and then relapsed after three months, and the third was a case of tinnitus without recruitment who was an immediate failure.

Slight residual function was found in three cases, indicating a good but not complete denervation. One case of Mèniére's disease was relieved permanently of vertigo and his hearing returned to normal, in another vertigo stopped for 18 months and then returned, and in the third case vertigo returned after 7 months.

Marked sympathetic activity was found in one case in which the first operation was an immediate failure; the hearing fell from 20 to 45 db. 18 months later. The opposite side was operated upon, and the patient was significantly improved, but large quantities of barbiturates are required to maintain freedom from attacks.

These results have no consistent pattern, so that the efficacy of the operation in relieving the symptoms cannot be directly related to the degree of sympathetic activity present in the upper limb.

TABLE 2.—*Assay of Residual Sympathetic Function Two or More Years Postoperatively*

Total number of cases examined	7
Cases with no residual sympathetic function	3
Cases with slight residual sympathetic function	3
Cases with good residual sympathetic function	1

Stellate Ganglion Block as a Diagnostic Tool

It was hoped that the use of preoperative procaine block of the stellate ganglion would give consistent information of prognostic value so that operation could be advised with considerable confidence.

At the outset of this study it was thought that the hearing was occasionally affected favorably by the temporary Horner's syndrome, but on reviewing these slight improvements of 5-15 db. in the light of present experience it would seem erroneous to attach significance to them. On no occasion was the hearing improved dramatically.

The most convincing evidence that there can be an important relationship between the sympathetic system and the diseased ear is that on three occasions I have stopped an attack of vertigo with a stellate block within five minutes of inserting the needle; each patient had a pronounced second degree or greater nystagmus, which vanished simultaneously with the vertigo. I know of no other way of stopping an acute attack. It might be said that the use of a 3 in. lumbar puncture needle constitutes good psychotherapy and controls the vertigo in this way; but the instant stopping of the nystagmus, which usually persists for two or three hours after the vertigo has subsided, is to me conclusive evidence that intervention in the the sympathetic system can have a favorable effect on Mèniére's disease.

It is customary to give a patient who is about to have 5-10 cc. of 1% procaine injected deeply a small quantity of one of the short-acting barbiturates to afford protection against a possible sensitivity reaction. This is permissible while determining the effect of the block on the circulation of the upper limb, for instance; but a barbiturate must not be used when trying to determine the effect on tinnitus or hearing. I have never used the barbiturate in this way, although I have soluble barbiturates ready for injection should the occasion arise.

Unless the patient states unequivocally that the tinnitus has stopped, no deduction

should be made concerning the efficacy of the block. Improvement or mere lessening of the tinnitus may be due to the desire of the patient to please you or the "woozy" sensation induced by the block on the head.

Seven cases had their tinnitus or vertigo stopped temporarily by procaine, and of these the subsequent operation was an initial success in five and an immediate failure in two.

Six cases were operated upon in spite of the fact that their tinnitus was unaffected by the block, and in these the operation was initially successful in two and an immediate failure in four.

From these few cases it would appear that a stellate block gives only slight information as to the outcome of the operation.

Summary

In summary I should like to make the following comments:

1. There can be no doubt that intervention in the sympathetic system can have an effect on the diseased labyrinth.

2. The only cases which are likely to demonstrate such a relation are those in which the pathology appears to be located in the labyrinth. Disease of central or middle ear origin is never affected by such intervention. All the cases that have demonstrated such a relationship have been found to have recruitment.

3. Out of 13 cases operated upon only 3 have remained completely free of symptoms for two or more years; 8 other patients regard themselves as successes because they are better than before operation, but because they continue to have attacks or require additional medication I classify them as failures. Only two patients regretted the operation, one of whom got a serious and troublesome wound infection and the other was an immediate and total failure and had to have a destructive labyrinthotomy.

4. Three successes out of 13 cases is less than the five successes out of 17 cases reported by Lewis in 1954, but it is in stark contrast with the results of Passe, who reported success in 60% of his cases.

5. This form of treatment is not the panacea it promised to be for all cases of labyrinthine disease and should only be considered in the following situations:

a. Intractable tinnitus which can be stopped by a stellate block.

b. Intractable Ménière's disease in which the opposite ear has been deafened by an associated condition, making labyrinthotomy impossible.

6. Four years ago it appeared that this approach to Ménière's disease would solve many difficulties; with the relapses came discouragement. The review has shown, I believe, that dorsal sympathectomy has but a small place in the treatment of these problems. The study, however, has been as fascinating as it has been disappointing.

Dr. Reginald H. Smithwick and his associates contributed freely from their vast experience with disorders of the sympathetic system and made this study possible. Dr. J. Edwin Wood evaluated the postoperative vascular studies.

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Congenital Atresia of the Middle and External Ear

ANDRES BUSTAMANTE GURRIA, M.D., and LEO DEUTSCH, M.D., México, D.F.

For diagnosis of congenital atresia we have the following resources at our disposal: radiology, for the morphologic analysis of the malformation; audiology, for functional classifications, and, in exceptional cases, otoscopy.

It is our routine procedure in the radiological study of each case to depend on three projections: the lateral of Schüller, the transversal of Stenvers, and the axial of Mayer. We believe, as opposed to House and Owen, that it is not sufficient to request a radiograph of the Stenvers type only. This certainly permits us to identify the epitympanum with the ossicles, and it also guides us concerning certain details of the labyrinth; but it leaves formations of great interest to the surgeon without the possibility of examination, such as the styloid process and the walls of the external auditory meatus, which are easily visible in a radiograph of the Mayer type. Nor can we neglect the Schüller projection due to the information which we obtain from it concerning the position of the lateral sinus, of the tegmen, and with reference to the pneumatization and the temporomandibular joint.

We agree that the fundamental purpose of a radiograph is that of providing the surgeon with preoperative data concerning the anatomical alterations which are to be found. Nevertheless, upon examining atresias radiologically, we have become aware of the fact that the picture leads us to interesting conclusions with respect to the evolutional mechanism of this malformation. Taking into consideration the limited number of autopsy findings, the roentgen picture represents an appropriate method for increasing our experience with respect to ontogenesis.

On the basis of the views expounded above, one of us (Deutsch), together with Mayer, attempted to classify atresias according to radiological findings. But experience obtained in 12 additional cases has led us today to reverse some of our former judgments. We also had to take the histological findings into consideration. These show that in the occlusion of the lumen of the external auditory meatus, apart from the malformed tympanic ring, not only the styloid process and the mastoid take part but also the temporal squama (Altmann), an alteration which we could definitely prove in some of our cases.

Our former conclusions and our present experiences resulted in the following outline, in which we distinguish two groups of atresias according to the state of the tympanic bone: on the one hand, hypoplastic cases and, on the other, the hyperplastic.

Group A: Hypoplasia of the tympanic bone
1. With substitution of the defect by
   (*a*) Hyperplastic styloid process
   (*b*) Excessively developed mastoid
   (*c*) Outgrowths formed from the squama
2. Without substitution of the defect

Group B: Hyperplasia of the tympanic bone

We cannot deny that the "pure" forms as mentioned above are rare. Our experience with our present series is that combinations of different types prevail.

Nevertheless, we have observed five cases which corresponded in their morphology to the conditions agreed upon by Alexander and Benesi, that is, that the hyperplastic upper end of Reichert's cartilage substitutes

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Fig. 1.—Mayer's projection: Right side: normal conditions. Left side: tympanic ring slightly hypoplastic. The styloid process excessively developed, filling almost completely the external meatus and partially the attic and aditus. *1*, head of the ramus; *2*, pyramid; *3*, external meatus; *4*, tympanic cavity on the right side and rudiments of aditus and attic on the left; *5*, labyrinth; *6*, antrum, absent on the left side; *7*, region of mastoid; *8*, outline of the mastoid; *9*, styloid process.

the defect originated by the hypoplastic tympanic bone.

In three cases, also of the "pure" type, the malformed tympanic bone acted as the only element in the formation of the atretic plate.

The remaining atresias were of the combined type (eight cases), without distinction

Fig. 2.—Mayer's projection: In this case the hypoplastic tympanic ring is visible, with its anterior (*aW*) and posterior wall (*pW*). The operative cavity (*Op*) and styloid process (*St pr*) excessively developed. The arrow indicates a defect of the anterior wall of the tympanic cavity.

Fig. 3.—Mayer's projection: The complete atresia (*A*) is formed by a hyperplasia of the tympanic ring. A small rest of the tympanic cavity, anterior part, persists (*TC*). *An*, antrum; *Ca*, carotic canal; *Lab*, labyrinth.

as to whether they appeared because of a defective tympanic bone or as the consequence of a hypertrophied membranous bone. In the first instance the substitution of the defect was accomplished not only by the styloid process but also by the temporal squama (three cases) (Fig. 4).

There were also cases without substitution of the defect (Fig. 5).

It is noteworthy that atresias exist where there is a combination of hypertrophy of the head of the styloid process and a hyperplastic tympanic bone. It is clear that this situation contradicts the postulation of Alexander and Benesi.

The two cases in which the atresia was formed by the hyperplastic tympanic bone and by an outgrowth of the squama recall those described by Altmann. But it is well to keep in mind the fact that it is not possible to differentiate between a hyperplastic tympanic ring and the upward process arising from the floor of the middle ear.

We do not propose to discuss the pathogenesis of atresias. We simply wish to point

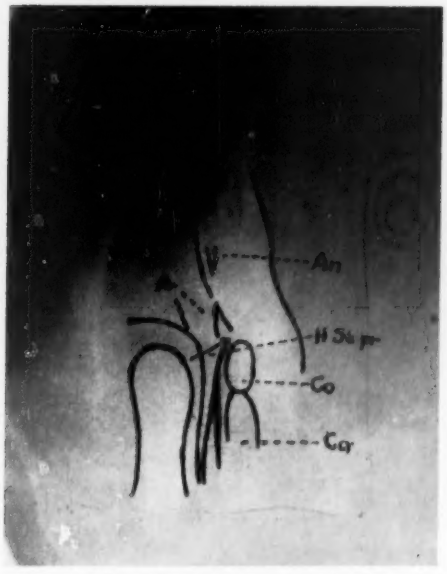


Fig. 4.—Mayer's projection: Combined form. The atresia is formed by a hypoplastic tympanic ring with thickened walls which close the external meatus (*A*). The head of the slightly hyperplastic styloid process completes the atresia (*H St pr*). The arrow indicates a defect of the anterior wall of the tympanic cavity. *An*, antrum; *Co*, cochlea; *Ca*, carotic canal.

out that in our series there were six cases with rather extensive pneumatization, a fact which speaks in favor of a "localized damaging factor which acts upon a circumscribed area only" (external meatus, tympanic cavity) without including the process of pneumatization (Altmann). Besides, it attacks genetically different structures, such as the temporomandibular joint, which we found deformed on two occasions.

In our cases there was no family history, which, with certain reservations, seems to discard the possibility of gene linkage.

The observations made in this series confirm that congenital atresia does not represent a definite and well-characterized entity. On the contrary, it is a polymorphic one. In the occlusion of the external auditory meatus there is a participation of the derivatives of the second branchial arch and adjacent structures of completely different genetic origin. We did not mention the changes which occurred in the elements of the first branchial arch, as these are not accessible to radiological examination. Later in the paper we will present examples of deformed ossicles.

For many years, the problem of congenital atresia of the middle and external ear has in most instances failed to be solved. On the basis that the function of the middle ear is a transmitter and amplifier of sound, it was logical to attempt its restoration by mechanical means. Failures discouraged the surgeons until advancements in surgery gave them new hope. With this spirit, in the last years, encouraged by the developments in grafts, fenestration, and, lately, tympanoplasty, they have made real progress in solving the problem. Those who are interested in the embryological problem ought to become more familiar with the marvelous process that takes place in the middle ear in the formation of a perfect acoustic instrument. Recent studies by Békésy, Weber, and Kobrak, among others, point out the complexity of this structure and orient us in the search of new routes directed toward solving the problem of congenital atresia.

The great otological surgeons of all times have made outstanding contributions in this field. Among the most recent contributors are Shambaugh, Baron, House, Wulstein, Zöllner, etc.

In principle, all established lesions that cause conductive hypacusia ought to be

Fig. 5.—Mayer's projection: Right side: normal conditions. Left side: complete aplasia of the tympanic ring, with the result that the head of the mandible, without support, leans into the region of the antrum. *1*, head of mandible; *2*, pyramid; *3*, external meatus; *4*, tympanic cavity; *5*, labyrinth; *6*, antrum; *7*, mastoid.

eliminated by the most reasonable means: surgery. We include here the atresias of the middle ear and the external auditory meatus.

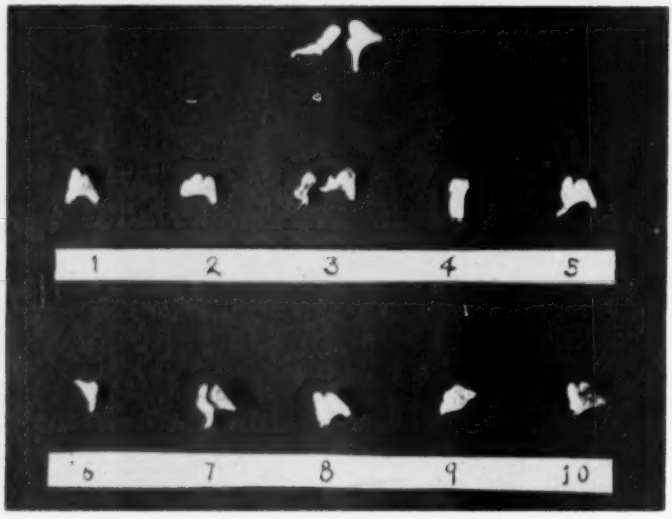
Audiophones should be indicated for cases considered unsuitable for surgery because of anatomical or other reasons.

In this article we have not included the subject of restoration of the auricle, because we have not had experience in this matter and consider it a subject pertaining more to plastic surgery than to otology.

In our experience the results obtained in the restoration of the auricle have not been satisfactory. In the majority of our cases, we decided to use an artificial auricle of plastic material. Although imperfect, it appears to us better than the results obtained by plastic surgery, more so because plastic surgery requires a large number of operations, with their inconveniences (psychological, economical, etc.) for the patient.

It is agreed that the intervention is more necessary in cases of bilateral atresia. One ought to wait to perform the operation until the ear has reached an appreciable development. For example, at the age of 10 to 12 years, or more, the operation is less difficult and one can obtain a more ample cavity.

In young girls when there exists some defect in the auricle, it can be hidden with the hair, whereas this is impossible in boys. Then, it is preferable in boys to use a plastic ear, to avoid in part the teasing of schoolmates.



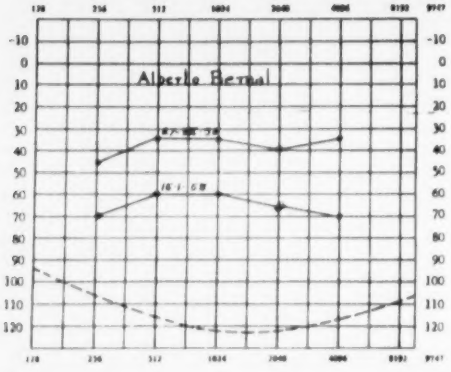
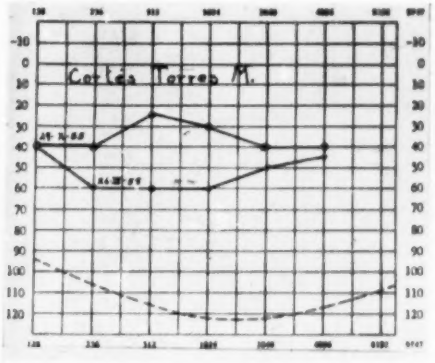
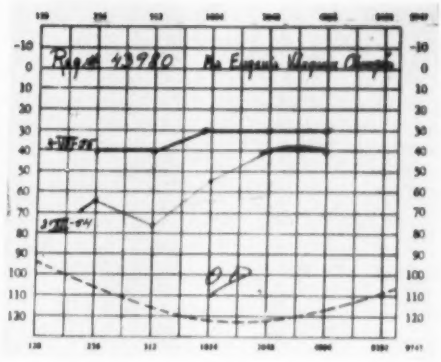


Fig. 6.—The malleus and incus, found to be joined and deformed, removed during operation in 10 cases.





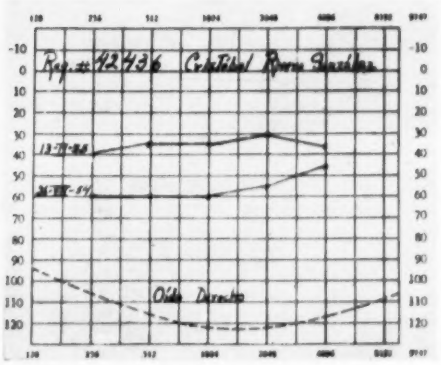


Fig. 7.—Three audiograms showing the improvements of hearing after surgical treatment. The lower line represents the preoperative level; the upper, the postoperative level.

In cases of unilateral atresia, where one ear is normal, the problem of audition or speech is not present; but in cases of bilateral atresia it is necessary to use audiophones until the patient reaches the stage where surgery can solve the problem. In general all atresias of the middle and external ear where clinically and radiologically there is an internal ear in good condition should be surgical cases.

From the operative point of view, we can distinguish two groups of patients: (1) cases where there is, more or less, pneumatization and (2) cases where pneumatization does not exist.

In the first group, a mastoidectomy is done and through this route the tympanic cavity is reached. Mastoidectomy is made as ample as possible. The external wall is removed together with the membrane that frequently occupies the place of the tympanum, also the incus and a portion of the malleus, which are almost always found joined, forming one unit, as seen in Figure 6. We respect the mucous membrane of the internal wall and the Eustachian tube. Once hemostasis is made, we check the mobility of the stapes. Whether this is mobile or not we apply a free graft, which should be in close contact with the stapes

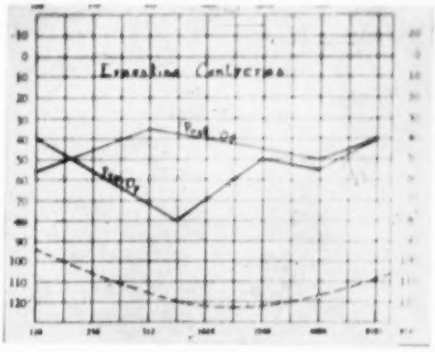


Fig. 8.—Two audiograms showing the improvement after fenestration. The upper line corresponds to the postoperative level and the lower to the preoperative level.

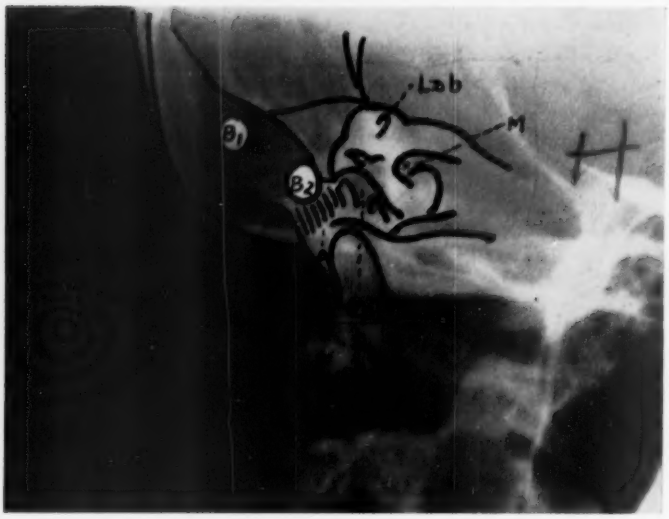


Fig. 9.—Attempt to locate the tympanic cavity by placing bullets in operative cavities. Stenvers' position. In this projection are seen the bullets, B_1 in the Citelli angle and B_2 in the zygomatic process. Between B_2 and the epitympanum (*Epi*) there still is a piece of bone, which belongs to atresia (*A*). *Lab*, labyrinth; *M*, internal meatus with cochlea and facial canal.

in order to form a new tympanic cavity. Next we cover the entire cavity with strips of skin. These strips should reach the outer portion of the future external auditory meatus. Over the strips of skin, layers of nylon or rayon are applied. Finally, we fill in the cavity with sponge and petrolatum (Vaseline) gauze.

It is important that the skin placed on the bottom amply cover the horizontal canal, especially if the stapes is not mobile, considering that a fenestration needs to be done. An ample cavity and auditory meatus should always be made. When pneumatization is more extensive, the operation is less difficult and has a better chance of success. In cases where the external auditory meatus exists, the endaural path in the Lempert manner seems the proper method, in our opinion. The graft is then applied over the stapes if it is mobile; if it is not, the fenestration is performed.

Fig. 10.—Same case as that shown in Figure 9. Schüller's projection: Bullet B_2 is placed high over the epitympanum. *1*, mastoid process; *2*, posterior root of zygoma; *3*, labyrinth; *4*, internal meatus; *5*, hypoplastic tympanic ring. Enlarging the operative cavity downward and medially, we opened the epitympanum, finding the ossicles.

The delayed application of the graft, as recommended by Guilford, may constitute a great advance in the technique.

In the second group, where pneumatization does not exist, the surgical problem is greater and the results, at least in our experience, are less satisfactory.

The localization of the tympanic cavity is almost always very small, and difficult; the technique described by House for these cases has been clearly demonstrated. The bone generally bleeds profusely, and the new path we have made is narrow and has a tendency to close. The danger of damaging the facial nerve is greater. In our radiographic studies can be seen the attempt to locate the tympanic cavity (Figs. 9 and 10).

In one of our most recent cases the tympanic cavity was found reduced. A small bone (probably the deformed incus) was discovered, while the stapes and the round window were impossible to identify. The tympanic end of the Eustachian tube was apparently normal.

The operative findings coincide in general with those described by other authors. The immobility of the stapes in both cases where we had to perform fenestration occurred in adults, and during the operation we were not able to determine the cause for the immobility.

Conclusions

1. A complete radiological examination of a patient with congenital ear atresia should include Mayer's, Stenvers' and Schüller's positions.

2. X-ray studies not only give information about pathogenesis but also orient the surgeons.

3. Fifty per cent of the cases correspond to the so-called "pure" forms, the other fifty per cent corresponds to the "polymorphic" group.

4. Most recent advances in surgical techniques render new possibilities for surgical treatment.

5. Final results depend mainly upon pneumatization and the condition of the tympanic cavity.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Hearing Rehabilitation Centers in the United States*

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The following listing of hearing centers in the United States was compiled from questionnaires sent to the centers, and returned during the period of Aug. 15, 1956, to Oct. 1, 1956.

When questionnaires were not returned, the information from the previous survey was used [1] and marked with an asterisk. Eighteen new centers were added to this year's listing, and nineteen formerly listed centers were dropped. Exclusion was made on the basis of inadequate equipment, dissolution of the center, or failure to reply to questionnaires since 1954.

The following is the key to abbreviations in the list and Table (x indicates that the item or service is offered).

Medical Supervision
- Oto. Otologist
  - C Consultant otologist
  - S Staff otologist
- Phys. Physician
  - C Consultant physician
  - S Staff physician

Referrals
- Med. Referrals from doctors
- Ex. Exclusive medical referrals
- P Partial medical referrals
- O Other referrals from nonmedical sources

Restrictions (on clientele accepted)
- V Restricted to veterans only
- VP Veterans and general public
- P General public
- H Hospital patients only
- C Children only

Personnel (certification in American Speech and Hearing Association)
- M Member
- F Fellow
- AH Advanced Hearing Certification
- AHP Application pending for Advanced Hearing Certification
- BH Basic Hearing Certification
- BHP Application pending for Basic Hearing Certification
- AS Advanced Speech Certification
- ASP Application pending for Advanced Speech Certification
- BS Basic Speech Certification
- BSP Application pending for Basic Speech Certification

Equipment
- Pure-Tone Aud. Pure-tone audiometers
- Speech Aud. Speech audiometers

Sound Rooms
- S Single sound room
- D Double sound room set-up
- PGSR Psychogalvanometer
- Special Other equipment, such as audio-delay unit, etc.

Services
- Pure-Tone Audio. Pure-tone audiometry
- Speech Audio. Speech audiometry
- H/A Selection Testing and fitting of hearing aids for the patient
- H/A Demon. Trial of hearing aids without testing
- Lipreading Classes in lipreading training
- Auditory Train. Classes in auditory training
- Speech Train. Speech improvement therapy
- Psych. Testing Psychometric testing
- Counsel. and Guid. Counseling and guidance in problems of hearing loss and parental counseling

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From the University of Denver (Mrs. Downs). Executive Director, The Audiology Foundation (Mrs. Carson).

* Special article.

Number of Center	Medical Supervision				Referrals			Restrictions	Personnel																				Equipment									Services			
	Oto.		Phys.		Med.		O		Total Number	Degrees					Certification														Pure-Tone Aud.	Speech Aud.	Sound Rooms		Special	Pure-Tone Audio.	Speech Audio.	H/A Selection	H/A Demon.	Lipreading	Auditory Train.	Speech Train.	Psych. Testing	Counsel. and Guid.	
															Audiology						Speech										FGSR										
	C	S	C	S	Ex.	P				B.A.	B.S.	M.A.	M.S.	Ph. D.	M	F	AH	AH P	BH	BH P	AS	AS P	BS	BS P			S	D												
1	x	..	..	..	..	x	x	VP	3	1	..	..	..	2	3	..	1	..	..	..	1	..	1	..	3	1	1	1	1	1	x	x	x	..	x	x	x	x	x	
2	x	..	x	..	..	x	x	VP	15	6	..	6	1	2	9	2	1	..	..	..	3	..	1	..	4	1	2	1	1	x	x	x	x	x	x	x	x	x	x	
3	x	..	x	..	..	x	x	P	1	..	..	..	..	1	1	..	..	..	1	..	..	..	1	..	1	1	..	..	x	..	..	x	x	x	..	x	x	x	x	x	
4	x	..	..	..	..	x	x	VP	1	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	1	1	1	1	1	..	x	x	x	..	x	x	..	..	..	
*5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
*6	x	..	..	..	..	x	x	P	3	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	1	..	..	..	..	x	..	x	..	..	..	x	x	x	
7	x	..	..	..	x	..	..	V	5	..	1	4	..	..	3	..	..	..	..	..	..	..	1	..	5	2	..	2	2	1	x	x	x	x	x	x	x	x	x	
8	x	..	..	..	x	..	..	C	2	..	..	1	1	..	2	1	1	..	..	..	..	..	1	..	3	1	2	1	1	..	x	x	x	x	x	x	x	x	x	
9	x	..	x	..	..	x	x	C	10	2	..	7	..	1	1	..	..	1	..	..	..	..	..	..	2	1	..	1	..	..	x	x	x	x	x	x	x	x	x	
10	x	..	..	..	..	x	x	P	17	11	..	5	..	3	17	1	..	..	..	1	3	..	5	..	3	1	2	1	1	2	x	x	x	x	x	x	x	x	x	
11	x	..	..	..	..	x	x	P	3	1	..	2	..	..	2	..	..	..	..	..	..	..	..	..	2	1	1	2	..	..	x	x	x	x	x	x	x	x	x	
*12	x	x	..	..	..	x	x	V	2	..	..	1	..	1	2	..	..	..	..	1	..	1	..	..	4	1	1	1	1	..	x	x	x	x	x	x	x	x	x	
13	x	..	..	..	x	..	..	P	12	6	..	2	..	4	6	1	2	..	..	..	1	..	..	..	3	2	..	2	1	..	x	x	x	x	x	x	x	..	x	
14	x	..	..	..	..	x	x	P	4	..	..	1	..	3	2	2	1	..	..	1	1	..	..	..	4	1	1	1	..	..	x	x	x	x	x	x	x	..	x	
15	x	..	x	..	..	x	x	P	2	..	..	..	..	2	2	..	..	..	2	..	1	..	1	..	1	..	1	..	..	..	x	..	..	..	x	x	x	x	x	
16	x	..	..	..	..	x	x	P	6	1	..	3	..	1	2	..	..	..	1	..	..	..	1	..	1	1	1	..	..	..	x	x	..	..	x	x	x	..	x	
17	x	..	..	..	..	x	x	VP	4	1	..	1	..	1	3	1	..	1	..	..	1	..	..	..	4	2	..	2	1	..	x	x	x	..	x	x	x	x	x	
18	x	..	..	..	..	x	x	P	4	1	1	..	..	..	1	..	..	..	..	..	..	..	..	..	2	1	2	..	..	..	x	x	x	..	x	x	x	..	x	
19	x	x	..	..	x	..	..	P	4	..	3	..	1	..	4	..	..	..	..	1	..	..	..	..	5	3	3	1	1	x	x	x	x	..	x	x	x	x	x	
20	x	..	..	..	..	x	x	VP	2	..	..	2	..	..	2	..	..	..	1	..	..	..	..	..	3	1	2	1	1	..	x	x	x	x	x	x	x	x	x	
21	..	x	..	..	x	..	..	V	24	9	..	7	..	6	10	..	1	..	1	..	..	..	3	..	10	4	4	4	1	x	x	x	x	x	x	x	x	x	x	
22	x	..	..	..	..	x	x	C	2	..	1	..	1	..	2	..	..	..	..	..	..	..	..	..	2	1	..	1	1	..	x	x	x	x	x	x	x	x	x	
23	x	..	..	..	x	..	..	P	3	..	..	2	..	1	2	..	1	..	1	..	..	..	1	..	3	2	2	..	1	..	x	x	x	x	x	x	x	x	x	
24	..	..	..	..	..	x	x	P	2	..	1	1	..	..	2	..	..	..	..	..	..	..	..	..	1	1	1	..	..	..	x	x	x	..	x	x	x	..	x	
25	x	..	..	..	..	x	x	P	3	..	..	2	..	..	1	..	..	1	..	..	..	..	..	..	2	1	..	1	..	..	x	x	x	x	x	x	x	..	x	
26	x	..	..	..	..	x	x	P	4	..	..	1	..	3	4	..	..	..	2	..	2	..	..	..	4	1	1	1	1	x	x	x	x	x	x	x	x	x	x	
27	..	x	..	..	x	..	..	V	2	..	..	2	..	..	2	..	..	..	1	..	1	..	..	..	4	2	..	2	1	1	x	x	x	x	x	x	x	..	x	
28	..	..	x	..	..	x	x	P	6	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	x	..	..	..	x	x	x	..	x	
29	..	..	..	x	..	x	..	V	1	..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	3	1	..	1	1	x	x	x	x	..	x	x	x	x	x	
30	x	..	x	..	..	x	x	P	6	3	1	2	..	..	4	2	1	..	..	..	2	..	..	..	1	1	1	..	..	..	x	x	x	..	..	x	x	x	x	
31	x	..	..	..	..	x	x	P	20	7	1	8	..	2	10	..	..	1	2	..	1	..	2	..	4	1	..	1	..	..	x	x	x	x	x	x	x	x	x	
32	x	..	..	..	..	x	x	VP	5	..	1	2	..	2	4	1	1	..	2	..	..	..	2	..	7	1	5	2	1	1	x	x	x	..	x	x	x	x	x	
33	..	x	..	..	x	..	..	V	5	..	..	5	..	..	4	..	..	..	1	..	..	..	1	..	8	4	3	3	2	x	x	x	x	x	x	x	x	x	x	
34	x	..	..	..	..	x	x	P	1	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1	1	1	..	..	x	x	x	x	..	x	x	x	..	..	
35	..	x	..	..	x	..	..	C	3	..	1	1	..	..	2	..	1	..	..	1	..	..	..	1	2	1	1	1	..	x	x	x	x	x	x	x	x	x	x	
36	..	x	..	x	x	..	..	P	6	..	..	3	..	..	1	..	..	..	1	..	..	..	1	..	1	1	1	..	..	..	x	x	x	..	..	..	x	x	..	
37	x	..	..	..	..	x	x	P	4	..	..	2	..	2	3	1	1	1	..	2	1	..	1	..	4	3	2	3	2	2	x	x	x	..	x	x	x	..	..	
38	..	x	..	..	..	x	x	P	1	..	..	1	..	..	1	..	..	..	1	..	..	..	1	..	1	1	..	1	..	..	x	x	x	x	x	x	x	x	x	
39	x	..	x	..	..	x	x	P	8	1	2	4	..	1	3	..	..	1	1	..	..	1	1	..	4	2	1	2	1	..	x	x	x	x	x	x	x	x	x	
40	..	x	..	x	..	x	x	P	4	..	1	2	..	..	3	..	1	..	..	..	..	..	1	..	2	1	1	1	1	x	x	x	x	x	x	x	x	x	x	
41	x	..	..	..	x	..	..	P	4	..	..	2	1	1	4	..	..	..	..	1	..	..	..	..	1	1	1	1	1	x	x	x	x	..	..	x	x	x	x	
*42	x	..	..	..	..	x	x	P	6	..	..	3	1	2	..	2	3	..	1	1	..	..	1	..	6	5	1	2	1	1	x	x	x	..	x	x	x	..	x	
*43	x	..	x	..	..	x	x	C	4	..	1	2	..	1	3	..	..	..	..	..	..	..	..	..	2	1	1	1	1	..	x	x	x	..	x	x	x	x	x	
44	x	..	..	x	..	x	x	P	1	..	..	..	..	1	1	..	1	..	..	..	..	..	..	..	1	1	1	1	1	..	x	x	x	..	x	x	x	x	x	
45	x	..	x	..	..	x	x	P	9	3	..	3	..	1	..	..	..	..	..	1	..	..	..	2	1	..	1	..	..	..	x	..	..	..	x	x	x	x	x	
46	x	..	x	..	..	x	x	P	3	..	..	2	..	1	3	..	..	..	..	..	..	..	3	..	3	..	1	..	..	..	x	..	..	..	x	x	x	..	..	
*47	..	x	..	x	..	x	x	P	9	2	2	2	..	3	4	1	..	..	1	..	1	..	3	..	11	1	..	..	..	..	x	x	x	x	x	x	x	x	x	
48	..	x	..	..	x	..	x	VP	6	1	..	4	1	..	6	..	2	..	..	1	..	..	2	..	8	3	..	3	1	..	x	x	x	x	x	x	x	x	x	
49	x	..	x	..	..	x	x	P	25	2	7	9	3	4	15	2	2	..	2	..	4	..	6	..	10	2	4	2	2	1	x	x	x	..	x	x	x	x	x	

* No information received for 1956 listing

Survey of Hearing Rehabilitation Centers in the United States—Continued

Number of Center	Medical Supervision				Referrals			Restrictions	Personnel																	Equipment							Services						
	Oto.		Phys.		Med.		O		Total Number	Degrees						Certification										Pure-Tone Aud.	Speech Aud.	Sound Rooms		VGSB	Special	Services						
																Audiology						Speech																	
	C	S	C	S	Ex.	P				B.A.	B.S.	M.A.	M.S.	Ph. D.	M	F	AH P	AH BH P	BH P	AS	AS P	BS	BS P			S	D			Pure-Tone Audio.	Speech Audio.	H/A Selection	H/A Demon.	Lipreading	Auditory Train.	Speech Train.	Psych. Testing	Counsel. and Guid.
50	..	..	x	..	..	x	x	P	1	..	..	..	1	..	1	..	..	..	..	..	..	..	1		4	1	2	..	..	..	x	x	x	x	x	x	x	x
51	..	..	..	..	..	x	x	P	2	..	..	..	1	1	2	..	..	..	..	..	..	1	..		3	..	1	1	..	..	x	x	x	x	x	x	x	x
52	x	..	..	..	..	x	x	P	4	2	..	..	1	..	1	..	..	..	1	..	..	..	..		3	1	..	1	..	..	x	x	x	x	x	x	..	x
53	x	..	..	..	..	x	x	P	9	..	..	2	..	7	9	4	1	..	..	..	4	..	3	..	4	1	3	1	1	..	x	..	..	x	x	x	x	x
54	..	x	..	..	..	x	x	VP	6	2	..	1	..	2	3	1	..	..	..	..	1	..	1	..	6	2	4	2	1	3	x	x	x	..	x	x	x	x
55	..	x	..	..	..	x	..	P	6	..	..	2	2	2	4	..	1	..	..	..	..	..	..	..	3	2	1	1	1	..	x	x	x	x	x	x	x	x
56	x	..	x	..	..	x	x	VP	23	13	..	8	..	2	7	1	1	..	..	..	1	..	..	..	1	1	1	1	1	..	x	x	x	x	x	x	x	x
57	x	..	x	..	x	..	..	VP	6	2	..	3	..	1	2	..	..	..	1	..	..	..	..	..	2	3	..	2	1	1	x	x	x	x	x	x	x	x
58	x	..	..	..	..	x	x	VP	7	2	..	1	1	2	5	..	..	..	1	..	1	..	2	..	2	1	..	2	1	..	x	x	x	x	x	x	x	..
59	x	..	..	..	..	x	x	P	2	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	1	..	1	..	x	x	x	x	x	x	x	x
60	..	..	..	..	..	x	x	P	3	..	..	2	..	..	1	..	..	..	..	..	..	1	..		2	1	..	1	..	..	x	x	x	x	x	x	..	x
61	..	x	..	..	..	x	x	VP	6	..	..	3	..	3	5	2	3	..	1	..	1	..	1	..	7	2	4	1	4	1	x	x	x	x	x	x	..	x
62	x	..	..	..	..	x	x	P	5	2	..	3	..	..	1	1	1	..	..	..	1	..	..	..	3	1	..	1	1	..	x	x	x	x	x	x	..	x
63	x	..	..	..	..	x	x	P	5	..	1	1	..	3	4	1	..	..	1	..	1	..	2	..	3	1	..	1	1	..	x	x	x	x	x	x	..	x
64	..	x	..	..	x	..	..	V	3	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	4	3	..	2	2	3	x	x	x	x	x	x	x	x
65	x	..	x	..	..	x	x	VP	4	..	..	4	..	..	3	..	..	..	1	..	2	..	..	..	1	..	1	..	1	..	x	x	..	x	x	x	x	x
66	..	x	..	..	..	x	x	P	5	2	..	..	1	1	2	..	..	..	1	..	..	1	..	..	4	1	2	1	1	..	x	x	x	x	x	x	x	x
67	x	..	x	..	..	x	x	P	3	..	..	3	..	1	2	1	..	..	..	..	2	..	1	1	1	1	..	..	1	..	x	x	x	x	x	x	x	x
68	..	x	..	..	..	x	x	P	2	..	1	..	..	1	1	..	..	..	..	..	..	1	..		4	1	2	2	1	..	x	x	..	x	..	..	x	x
69	x	..	..	..	..	x	x	P	4	1	1	1	..	..	..	..	..	..	..	..	..	..	..	..	2	1	1	..	..	..	x	x	x	x	x	x	x	x
70	x	..	..	..	..	x	x	P	3	2	..	1	..	..	2	1	..	..	..	..	1	..	..	..	2	1	..	1	..	..	x	x	x	x	x	x	x	x
71	..	..	x	..	..	x	x	P	11	..	..	..	7	4	10	1	..	..	1	..	2	..	6	..	5	1	1	1	1	..	x	x	x	x	x	x	x	x
72	..	..	..	..	..	x	x	P	4	1	..	3	..	..	3	..	..	..	..	..	..	..	1	..	1	1	..	1	..	..	x	x	x	x	x	x	..	..
73	..	x	..	..	..	x	x	P	2	..	..	..	..	2	2	..	1	1	1	..	..	..	..	..	3	1	1	1	1	1	x	x	x	x	x	x	..	x
74	x	..	..	..	..	x	x	VP	14	1	..	5	5	3	12	1	1	..	1	..	1	..	..	..	5	3	3	2	1	..	x	x	x	x	x	x	x	x
75	..	..	..	..	..	x	x	P	5	..	..	..	1	4	5	..	..	..	..	..	1	..	..	..	4	1	3	1	..	..	x	x	x	x	x	x	x	x
76	..	..	..	..	..	x	x	C	3	..	..	2	..	..	1	..	..	..	1	..	..	..	..	..	2	..	1	1	1	..	x	x	x	x	x	x	..	x
77	x	..	..	..	..	x	x	P	2	..	..	1	1	..	2	..	1	..	..	..	..	..	..	..	3	1	..	1	1	..	x	x	x	x	x	x	x	x
78	x	..	..	..	..	x	x	P	5	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	1	..	..	x	x	..	x	x	x	..	x
79	x	..	..	..	..	x	x	C	21	4	..	6	..	..	4	..	3	..	..	..	..	..	..	..	46	7	..	3	..	..	x	x	..	x	x	x	..	x
80	..	x	..	..	x	..	..	VP	3	1	..	1	..	1	2	..	1	..	..	2	..	..	..	..	5	1	2	1	1	..	x	x	x	x	x	x	x	x
81	x	..	..	..	..	x	x	P	7	1	..	2	..	4	5	1	..	..	..	..	1	..	3	..	4	..	1	..	..	..	x	x	..	..	x	x	x	x
*82	x	..	..	..	..	..	..	H	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	4	1	x	..	x	x	x	x	x	x	x	x	x	x
83	..	..	..	..	..	x	x	P	3	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	4	1	..	..	..	..	x	x	x	..	x	x	..	..
84	x	..	..	..	x	..	..	V	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	2	1	2	1	1	1	x	x	x	x	x	x	..	x
85	x	..	..	..	..	x	x	VP	21	1	9	5	3	2	..	..	1	..	..	..	..	..	..	..	6	3	3	3	1	1	x	x	x	..	x	x	x	x
86	..	..	x	..	..	x	x	VP	7	..	..	4	..	3	7	..	2	..	..	..	3	1	1	..	4	1	..	1	1	1	x	x	x	x	x	x	x	x
87	..	x	..	..	..	x	x	P	3	2	..	1	..	..	1	..	..	..	..	1	1	..	..	..	5	1	1	1	1	..	x	x	x	x	x	x	x	x
*88	..	x	..	x	..	x	x	P	4	..	..	3	1	..	2	..	..	..	..	..	1	..	1	1	1	1	..	..	1	..	x	x	x	x	x	x	x	x
89	x	..	..	..	..	x	x	P	1	1	..	..	..	..	1	..	..	..	1	..	..	..	1	..	2	1	4	1	1	1	x	x	x	x	x	x	x	x
90	x	..	..	..	x	..	..	VP	3	..	..	..	1	..	1	..	..	..	1	..	..	..	..	..	2	1	1	1	1	1	x	x	x	..	..	..	..	..
91	..	x	..	x	x	..	..	C	3	..	..	..	3	..	2	..	..	..	1	..	..	..	..	..	2	1	1	1	1	..	x	x	x	..	x	x	x	x
92	..	x	..	..	x	..	..	V	7	..	..	5	..	1	3	..	..	..	1	..	..	..	2	..	6	3	3	3	2	2	x	x	x	x	x	x	x	x
93	..	x	..	..	x	..	..	P	7	6	..	5	..	..	7	..	..	..	1	..	1	..	1	..	6	2	2	2	1	1	x	x	x	x	x	x	x	x
94	x	..	..	..	..	x	x	P	4	..	..	4	..	..	4	..	..	..	..	..	1	..	2	..	2	1	1	1	..	..	x	x	x	x	x	x	x	x
95	..	x	..	..	..	x	x	P	4	1	..	3	..	..	3	..	..	..	..	..	..	..	2	..	3	3	1	1	1	..	x	x	x	..	x	x	x	x
96	x	..	..	..	..	x	x	P	6	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	9	2	3	3	..	1	x	x	x	x	..	..	..	..
97	..	x	..	x	..	x	x	P	5	..	..	3	..	2	4	..	..	1	..	..	1	..	..	..	4	1	2	1	1	..	x	x	x	x	x	x	x	x
98	x	..	x	..	..	x	x	P	13	5	..	6	..	..	2	..	1	..	..	..	..	..	1	..	4	2	2	..	1	..	x	x	x	x	x	x	x	x
99	x	..	..	..	..	x	x	P	1	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	3	..	1	..	..	..	x	..	x	x	x	x	x	x
100	..	x	..	..	..	x	x	P	3	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	3	2	3	1	1	..	x	x	x	x	x	x	..	..

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The sponsorship of the 145 centers can be broken down as follows:

Universities	42
Medical schools or hospitals	39
Hearing societies	26
Federal agencies	12
Public agencies	12
State agencies	9
Private	4

Listing in this survey does not constitute endorsement by any organization for the services rendered by the center. Any changes or corrections in listing should be sent to The Audiology Foundation, Box 21, Glenview, Ill.

Detailed information in regard to hearing rehabilitation centers listed below may be found in the Table.

Established Hearing Rehabilitation Centers

ALABAMA

1. Birmingham, Hearing and Speech Clinic, 704 18th St. S.
   Auspices: Medical College of Alabama
   Administrative Head: Robert E. Roach, Ph.D., AH
   Medical Supervisor: Frank S. Moody, M.D.
2. Tuscaloosa, Speech and Hearing Rehabilitation Center, P.O. Box 1965, University of Alabama
   Auspices: University of Alabama
   Administrative Head: T. Earle Johnson, Ph.D., AH
   Medical Supervisor: University Hospital and Medical College of Alabama Staff

ARIZONA

3. Phoenix, Speech and Hearing Center, 2021 N. Central Ave.
   Auspices: Private
   Administrative Head: Norman E. Iverson, Ph.D., BH, BS
   Medical Supervisor: Otolaryngologists from Maricopa County Medical Society
4. Phoenix, Samuel Gompers Memorial Clinic, 7211 N. Seventh St.
   Auspices: Maricopa County Society for Crippled Children and Adults
   Administrative Head: Richard Piper
   Medical Supervisor: Medical advisory council of local otologists

ARKANSAS

*5. Little Rock, Audiology Clinic, Veterans Administration Regional Office, 555 Building, 211 Broadway
   Auspices: Veterans Administration

CALIFORNIA

*6. Alhambra, Children's Hearing and Speech Clinic, 612 W. Shorb
   Auspices: National Auricular Foundation, Inc.
   Administrative Head: Willard B. Hargrave, Executive Vice-President
   Medical Supervisor: M. E. Trainor, M.D.
7. Los Angeles, Audiology and Speech Correction Clinic, Veterans Administration, 1031 S. Broadway
   Auspices: Veterans Administration
   Administrative Head: Edward W. Gibbons, M.A.
   Medical Supervisor: Harold Boyd, M.D., Consultant
8. Los Angeles, Hearing and Speech Clinic, 4614 Sunset Blvd.
   Auspices: Children's Hospital
   Administrative Head: Jacqueline Keaster, M.S., AH
   Medical Supervisor: Victor Goodhill, M.D.
9. Los Angeles, John Tracy Clinic, 806 W. Adams Blvd.
   Auspices: Nonprofit corporation
   Administrative Heads: Mrs. Spencer Tracy, Director; Edgar L. Lowell, Ph.D., Administrator
   Medical Supervisor: Victor Goodhill, M.D., Consultant

*No information received for 1956 listing.

10. Los Angeles, Speech-Hearing Clinic, University of Southern California, University Park
    Auspices: University of Southern California
    Administrative Head: Lee E. Travis, Ph.D., AS, F
    Medical Supervisor: Victor Goodhill, M.D.
11. San Diego, San Diego Hearing Society, 3843 Herbert St.
    Auspices: San Diego Community Chest
    Administrative Head: John Y. Miller, M.A.
    Medical Supervisors: John L. Feldman, M.D., and John Hendrickson, M.D.
*12. San Francisco, Audiology and Speech Correction Clinic, Veterans Administration Hospital, 42nd and Clement St. (Fort Miley)
    Auspices: Veterans Administration
    Administrative Head:
    Medical Supervisor: Walter P. Work, M.D., Consultant
13. San Francisco, San Francisco Hearing and Speech Center, 1609 Scott St.
    Auspices:
    Administrative Head: Kenneth O. Johnson, Director, Ph.D., AH
    Medical Supervisor: Committee of otologists (Robert C. McNaught, M.D., Chairman)
14. Stanford, Stanford Speech and Hearing Clinic, Stanford University
    Auspices: Stanford University
    Administrative Heads: Virgil A. Anderson, Ph.D., AS; Hayes A. Newby, Ph.D., AH
    Medical Supervisor: Community advisory board of physicians
15. Stockton, Speech and Hearing Center, College of the Pacific, Pacific Ave.
    Auspices: College of the Pacific
    Administrative Head: Howard L. Runion, Ph.D., AS
    Medical Supervisor: James Fischer, M.D.

COLORADO

16. Denver, Denver Hearing Society, 1556 Emerson St.
    Auspices: Community Chest Agency
    Administrative Head: Elizabeth Kaho, Ph.D.
    Medical Supervisor: Committee of doctors
17. Denver, Hearing and Adult Speech Center, University of Denver
    Auspices: University of Denver
    Administrative Head: Elwood Murray, Ph.D., F
    Medical Supervisor: Committee of otologists

CONNECTICUT

18. Hartford, Hartford Hearing League, Inc., 252 Asylum St.
    Auspices: Greater Hartford Community Chest
    Administrative Head: Mrs. Helen S. Lowry, Executive Director, B.A.
    Medical Supervisor: Consulting otologists
19. New Haven, Hearing and Speech Center, Yale-New Haven Medical Center, 789 Howard Ave.
    Auspices: Grace-New Haven Community Hospital and Yale School of Medicine
    Administrative Head: Harris I. Pomerantz, M.S., BSP, BHP
    Medical Supervisor: John A. Kirchner, M.D.

DELAWARE

20. Wilmington, Delaware Hospital Audiology and Speech Center, 501 W. 14th St.
    Auspices: Private general hospital
    Administrative Head: L. LeRoy Horne, M.A., BH
    Medical Supervisor: Edgar N. Johnson, M.D.

DISTRICT OF COLUMBIA

21. Washington, D. C., Audiology and Speech Center, Forest Glen Section
    Auspices: Walter Reed Army Hospital, Walter Reed Army Medical Center
    Administrative Head: James P. Albrite, Major (MC), M.D.
    Medical Supervisor: James P. Albrite, Major (MC), M.D.
22. Washington, D. C., Hearing and Speech Clinic, 2125 13th St., N.W.
    Auspices: The Children's Hospital of the District of Columbia
    Administrative Head: Mrs. Edna K. Monsees, B.S., M
    Medical Supervisor: Alan E. Walker Jr., M.D.

23. Washington, D. C., Leroy L. Sawyer Hearing and Speech Clinic, 1147 15th St., N.W.
Auspices: Episcopal Eye, Ear and Throat Hospital
Administrative Head: Mrs. Lorraine A. Roblee, M.A. BH, AS
Medical Supervisor: T. Frasier Williams, M.D.
24. Washington, D. C., Hearing Rehabilitation Center, 1911 R St., N.W.
Auspices: Private
Administrative Head: Mrs. Edna K. Monsees, B.S., M
Medical Supervisor: Referring otologists
25. Washington, D. C., Washington Hearing Society, 1934 Calvert St., N.W.
Auspices: United Fund
Administrative Head: Ruth Allison Hudnut, M.A.
Medical Supervisor: Group of otolaryngologists, consultants

FLORIDA

26. Gainesville, Speech and Hearing Clinic, University of Florida
Auspices: University of Florida Center of Clinical Services
Administrative Head: McKenzie Buck, Ph.D., AS
Medical Supervisor: Dekle Taylor, M.D.
27. Pass-A-Grille (St. Petersburg), Audiology and Speech Correction Clinic, Veterans Administration Regional Office
Auspices: Veterans Administration
Administrative Head: R. Donald Bitler, Chief, M.A., M
Medical Supervisor: August E. Gehrke, M.D.
28. St. Petersburg, Sertoma Speech Correction Clinic, American Legion Hospital for Crippled Children
Auspices: Sertoma Club
Administrative Head: R. Donald Bitler, M.A., M
Medical Supervisor: Members of Advisory Board

GEORGIA

29. Atlanta, Veterans Administration Audiology and Speech Correction Clinic, 105 Pryor St., N.E.
Auspices: Veterans Administration
Administrative Head: Allan J. Heffler, Chief, M.A., BH
30. Atlanta, Davison School of Speech Correction, 1780 N. Decatur Rd., N.E.
Auspices:
Administrative Head: Louise D. Davison, Director, B.A., AS
Medical Supervisors: Calhoun McDougal, M.D.; Lester Brown, M.D., and others
31. Atlanta, Junior League School for Speech Correction, Inc., 2020 Peachtree Rd., N.W.
Auspices: Junior League of Atlanta
Administrative Head: Claude S. Hayes, Acting President, Ph.D., AHP
Medical Supervisors: Lester Brown, M.D.; B. Russell Burke, M.D.; William Warren, M.D.

ILLINOIS

32. Champaign, University Hearing Center, 321 Illini Hall
Auspices: Department of Speech, University of Illinois
Administrative Head: E. Thayer Curry, Ph.D., AH
Medical Supervisor: Area otologists serve as consultants
33. Chicago, Audiology Clinic, Veterans Administration West Side Hospital, 820 S. Damen
Auspices: Veterans Administration
Administrative Head: Emory O. Windrem, M.A., BS
Medical Supervisor: Nathaniel Van Dorf, M.D.
34. Chicago, Chicago Hearing Society, 30 W. Washington St.
Auspices: Voluntary Health Agency; partial support from Chicago Community Fund
Administrative Head: Mary L. Thompson, M.A., Executive Director
Medical Supervisor: Consulting board of otologists
35. Chicago, Children's Memorial Audiology Clinic, 707 Fullerton Ave.
Auspices: The Children's Memorial Hospital
Administrative Head: Miss Alice Saar, Director of Clinics
Medical Supervisor: George S. Livingston, M.D.

36. Chicago, Hearing and Speech Service, 29th and S. Ellis Ave.
    Auspices: Michael Reese Hospital Medical Center
    Administrative Head: Peter A. Campanelli, M.A.
    Medical Supervisor: Robert Henner, M.D.
37. Chicago, Northwestern University Hearing Clinic, 303 E. Chicago Ave.
    Auspices: Department of Otolaryngology, Medical School
    Administrative Head: Raymond Carhart, Ph.D., AH, AS
    Medical Supervisor: George E. Shambaugh Jr., M.D.
38. Chicago, Speech and Hearing Center, 1753 W. Congress St.
    Auspices: Presbyterian Hospital of the City of Chicago
    Administrative Head: Duncan R. C. Scott, M.A., BS, BH
    Medical Supervisor: Stanton A. Friedberg, M.D.
39. Chicago, Illinois Speech and Hearing Center, 904 W. Adams St.
    Auspices: Illinois Eye and Ear Infirmary, University of Illinois College of Medicine
    Administrative Head: Charles Lightfoot, Ph.D., ASP, AHP
    Medical Supervisor: Richard A. Buckingham, M.D.
40. Chicago, St. Luke's Speech and Hearing Service, 1439 S. Michigan
    Auspices: St. Luke's Hospital
    Administrative Head: William F. Waldrop, M.A., AH
    Medical Supervisor: Aldert H. Andrews Jr., M.D.
41. Chicago, Speech Clinic, University of Chicago Audiology Service, 950 E. 59th St.
    Auspices: University of Chicago
    Administrative Heads: Joseph M. Wepman, Ph.D., F, AS; Ralph Naunton, Ph.D.
    Medical Supervisor: John Lindsay, M.D.
*42. Evanston, Northwestern University Hearing Clinics, Adult
    Auspices: Northwestern University
    Administrative Head: John H. Gaeth, Ph.D., AH
    Medical Supervisors: George E. Shambaugh Jr., M.D.; Eugene Derlacki, M.D.
*43. Evanston, Children's Hearing and Aphasia Clinic, Speech Annex, Northwestern University
    Auspices: School of Speech, Northwestern University
    Administrative Head: Helmer R. Myklebust, Ed.D., Director
    Medical Supervisor: George E. Shambaugh Jr., M.D.
44. Normal, Hearing Laboratory, Illinois State Normal University
    Auspices: State of Illinois, Illinois State Normal University
    Administrative Head: Glenn Taylor, Ph.D., AH
    Medical Supervisor: C. W. Hardy, M.D.
45. Peoria, Bradley University Speech Clinic, 815 N. Glenwood
    Auspices: Bradley University
    Administrative Head: Clara K. Mawhinney, Ph.D., AS
    Medical Supervisors: R. Hart, M.D.; M. Cohen, M.D., Consultants
46. Rock Island, Augustana College Speech and Hearing Center, 3500
    Auspices: Augustana College
    Administrative Head: Martin J. Holcomb, M.A., BS
    Medical Supervisor: Marvin Lasky, M.D.; John Burgess, M.D., Consultants

INDIANA

*47. Bloomington, Indiana University Speech and Hearing Center, University Clinics Building
    Auspices: Indiana University
    Administrative Head: Robert Milisen, Ph.D., F, AS
    Medical Supervisor: Bryan Quarles, M.D.
48. Indianapolis, Audiology and Speech Clinic, Indiana University Medical Center
    Auspices: Department of Otolaryngology and Bronchoesophagology, Indiana University Medical Center
    Administrative Head: Francis L. Sonday, A.B., AH
    Medical Supervisors: Marlow W. Manion, M.D.; David E. Brown, M.D.
49. Lafayette, Purdue University Speech and Hearing Clinic
    Auspices: Purdue University
    Administrative Head: M. D. Steer, Ph.D., F, AS
    Medical Supervisors: R. Calvert, M.D.; E. L. Van Buskirk, M.D., Consultants

50. Muncie, Ball State Teachers College Out-Patient Clinic
Auspices: Ball State Teachers College
Administrative Head: Alan Huckleberry, Ph.D.
Medical Supervisor: W. G. Pippenger, M.D.
51. Terre Haute, Indiana State Teachers College, Special Education Clinic
Auspices: Indiana State Teachers College
Administrative Head: Rutherford B. Porter, Ed.D.
Medical Supervisor: Floyd Riggs, M.D.

IOWA

52. Des Moines, Des Moines Hearing and Speech Center, 615 Locust St.
Auspices: Des Moines Community Chest
Administrative Head: William K. Ickes, M.S., BH
Medical Supervisor: Advisory committee of otologists
53. Iowa City, University of Iowa Speech and Hearing Clinic, East Hall
Auspices: University of Iowa
Administrative Head: James F. Curtis, Ph.D., F, AH
Medical Supervisor: D. M. Lierle, M. D.
54. Iowa City, Department of Otolaryngology, University Hospitals
Auspices: State University of Iowa College of Medicine
Administrative Head: D. M. Lierle, M.D., M.S., D.Sc., F
Medical Supervisors: D. M. Lierle, M.D.; C. M. Kos, M.D.

KANSAS

55. Kansas City, University of Kansas Medical Center, 39 and Rainbow
Auspices: University of Kansas Medical Center Board of Regents
Administrative Head: June Miller, M.S., M
Medical Supervisor: G. O. Proud, M.D.
56. Wichita, Institute of Logopedics, 2400 Jardine Dr.
Auspices: University of Wichita
Administrative Head: Martin F. Palmer, Sc.D, F, AS
Medical Supervisor: C. T. Hinshaw, M. D.

KENTUCKY

57. Lexington, Audiology Clinic, University of Kentucky
Auspices: Department of Psychology and Lexington Council of Jewish Women
Administrative Head: Frank Kodman Jr., Ph.D., BH
Medical Supervisor: Medical Advisory Committee

LOUISIANA

58. New Orleans, Tulane Speech and Hearing Center, 1430 Tulane Ave.
Auspices: Tulane University Medical School
Administrative Head: Jeannette K. Laguaite, Ph.D., AS
Medical Supervisor: James W. McLaurin, M.D.

MAINE

59. Portland, Portland Hearing Society, Inc., 723A Congress St.
Auspices: Private social agency with United Fund support
Administrative Head: Pauline G. Carter, B.A.
Medical Supervisor: None
Medical Supervisor: C. A. Almand, M.D.

MARYLAND

60. Baltimore, The Baltimore Hearing Society, Inc., 928 N. Charles St.
Auspices: Community Chest of Baltimore
Administrative Head: Mr. Jay Cherry, M.A., BS
Medical Supervisor: None
61. Baltimore, Hearing and Speech Center, Johns Hopkins Hospital
Auspices: Johns Hopkins School of Medicine
Administrative Head: William G. Hardy, Ph.D., AH
Medical Supervisor: John E. Bordley, M.D.

MASSACHUSETTS

62. Boston, Boston Guild for the Hard of Hearing, 283 Commonwealth Ave.
Auspices: United Community Services
Administrative Head: Mrs. Claire K. Kennedy, Executive Director
Medical Supervisor: Committee of consulting otologists supplied by Otolaryngological Association

63. Boston, Boston University Speech and Hearing Center, 332 Bay State Rd.
Auspices: Boston University
Administrative Head: Wilbert L. Pronovost, Ph.D., AS
Medical Supervisor: Four consultant otolaryngologists

64. Boston, Veterans Administration Audiology Clinic, 17 Court St.
Auspices: Veterans Administration
Administrative Head: George E. Hill Jr., M.A.
Medical Supervisor: Benjamin Bell, M.D.

65. Boston, Robbins Speech and Hearing Clinic of Emerson College, 145 Beacon St.
Auspices: Emerson College
Administrative Head: Catherine C. Perry, M.A. AS
Medical Supervisor: John Homans Jr., M.D.

66. Boston, Hearing and Speech Clinic, 300 Longwood Ave.
Auspices: Children's Medical Center
Administrative Head: Adam J. Sortini, Ed.D., BS, BH
Medical Supervisor: Medical Board (Chairman: Dr. Flake)

67. Boston, Institute for Speech Correction, 419 Boylston St.
Auspices: Board of Trustees
Administrative Head: Samuel D. Robbins, L. H. D., AS
Medical Supervisor: Gale C. Perry, M. D.

68. Boston, The Frederic Winthrop Foundation at the Massachusetts Eye and Ear Infirmary, 243 Charles St.
Auspices: Massachusetts Eye and Ear Infirmary
Administrative Head: Alan S. Feldman, Ph.D., BS
Medical Supervisor: Philip E. Meltzer, M.D.

69. Springfield, Springfield Hearing League, Inc., 1694 Main St.
Auspices: United Fund, Inc.
Administrative Head: Mrs. Grace F. Gaffney, A.B.
Medical Supervisor: Medical Advisory Board

70. Worcester, Worcester Hearing League, Inc., 306 Main St.
Auspices: Worcester Community Chest
Administrative Head: Miss Kathleen F. Kirby, B.A., Executive Director
Medical Supervisor: Board of three consultant otologists

MICHIGAN

71. Ann Arbor, University of Michigan Speech Clinic, 1007 E. Huron St.
Auspices: University of Michigan Speech Department and Institute for Human Adjustment
Administrative Head: H. Harlan Bloomer, Ph.D. AS, F
Medical Supervisor: Hsi-Yen Liu, M.D.

72. Detroit, Detroit League for the Handicapped, 1401 Ash
Auspices: United Community Services
Administrative Head: Robert W. Will, M.Ed.
Medical Supervisor: None

73. Detroit, Henry Ford Hospital Audiology and Speech Clinic, W. Grand Blvd. at Hamilton
Auspices: Henry Ford Hospital
Administrative Head: A. Bruce Graham, Ph.D., AHP
Medical Supervisor: J. Lewis Dill, M.D.

74. Detroit, Wayne State University Speech and Hearing Clinic, 656 W. Warren
Auspices: Wayne State University
Administrative Head: George A. Kopp, Ph.D., F., AS
Medical Supervisor: Heinrich Kobrak, M.D., Ph.D, Consultant

75. East Lansing, Speech and Hearing Clinic, Department of Speech
Auspices: Michigan State University
Administrative Head: Charles Pedrey, Ph.D., AS
Medical Supervisor: None

76. Flint, Michigan School for the Deaf (Hearing Clinic), Court St.
Auspices: State Board of Education
Administrative Head: Thomas H. Poulos, M.A., BH
Medical Supervisor: None

77. Kalamazoo, Constance Brown Society for Better Hearing, 316 Commerce Building
Auspices: Kalamazoo Foundation; Kalamazoo Community Chest
Administrative Head: Alfred R. Thea, M.S., AH
Medical Supervisor: Ralph B. Fast, M.D., Consultant

78. Lansing, Michigan Association for Better Hearing, 408 Hollister Building
Auspices: Michigan United Fund
Administrative Head: Stahl Butler, Executive Director, M.A.
Medical Supervisor: Seven otolaryngologists and two nurses on Board of Directors and Advisory Board

79. Lansing, Hearing Conservation Section, Michigan Department of Health, DeWitt Rd.
Auspices: Maternal and Child Health Division, Michigan Department of Health
Administrative Head: Courtney D. Osborn, Chief, Hearing Conservation Section, M.A., AH
Medical Supervisor: Goldie B. Corneliuson, M.D., and Subcommittee on Hearing Defects, Michigan State Medical Society

MINNESOTA

80. Minneapolis, Audiology Clinic, University of Minnesota Hospitals
Auspices: University of Minnesota
Administrative Head: Frank M. Lassman, Ph.D., AH
Medical Supervisor: Lawrence R. Boies, M.D.

81. Minneapolis, Speech and Hearing Clinic, University of Minnesota
Auspices: University of Minnesota
Administrative Head: Ernest H. Henriksen, Ph.D., AS
Medical Supervisor: Donald Coman, M.D.

*82. Rochester, Mayo Clinic Audiology Center, EENT Department
Auspices: Mayo Clinic
Administrative Head: Henry S. Williams, M.D.
Medical Supervisor: Henry S. Williams, M.D.

MISSOURI

83. Columbia, University of Missouri Speech and Hearing Clinic, 325 Switzler Hall
Auspices: University of Missouri
Administrative Head: Charlotte G. Wells, Ph.D., AS
Medical Supervisor: None

84. Kansas City, Veterans Administration Regional Office, 1828 Walnut
Auspices: Veterans Administration
Administrative Head: Morton Zarcoff, M.S.
Medical Supervisor: A. L. Kelly, M.D.

85. St. Louis, Central Institute for the Deaf, 818 S. Kingshighway
Auspices: Central Institute for the Deaf, a Missouri Corporation
Administrative Head: S. Richard Silverman, Ph.D., AH
Medical Supervisors: Theodore Walsh, M.D.; Harold Cutler, M.D.

NEBRASKA

86. Lincoln, Speech and Hearing Laboratories, University of Nebraska
Auspices: University of Nebraska
Administrative Head: John Wiley, Ph.D., AS
Medical Supervisor: None

NEW JERSEY

87. Newark, Henry C. Barkhorn Memorial Hearing and Speech Center, 77 Central Ave.
Auspices: Newark Eye and Ear Infirmary

Administrative Head: Herbert E. Rickenberg, M.A., AS, BHP
Medical Supervisor: William H. Hahn, M.D.

*88. Newark, St. Barnabas Hearing and Speech Center, 701 High St.
Auspices: Hospital of St. Barnabas and for Women and Children
Administrative Head: Lyndon A. Peer, M.D.
Medical Supervisors: Franklin Keim, M.D.; Louise Fischer, M.D.

NEW MEXICO

89. Santa Fe, Hearing Test and Child Study Center, 1060 Cerrillos Rd.
Auspices: New Mexico School for the Deaf
Administrative Head: Jack A. Willeford, B.A., BH, BS
Medical Supervisor: Undetermined

NEW YORK

90. Buffalo, Buffalo Hearing and Speech Center, Inc., 2183 Main St.
Auspices: Buffalo Otolaryngological Society
Administrative Head: Elmo L. Knight, M.S., BH
Medical Supervisor: Hermann Bozer, M.D.

91. Buffalo, Children's Hospital of Buffalo Hearing and Speech Clinic, Bryant St
Auspices: Children's Hospital of Buffalo
Administrative Head: Elmo L. Knight, M.S., BH
Medical Supervisor: Mitchel Rubin, M.D.

92. New York, Audiology Clinic, 252 Seventh Ave.
Auspices: Veterans Administration
Administrative Head: Martin Cohen, M.A., BS, Acting Chief Audiologist
Medical Supervisor: Morris F. Heller, M.D.

93. New York, Columbia-Presbyterian Medical Center, Hofheimer Speech and Hearing Clinic, 622 W. 168th St.
Auspices: The Presbyterian Hospital of the City of New York
Administrative Head: Maurice H. Miller, M.A., AS, AHP
Medical Supervisor: Edmund P. Fowler Jr., M.D.

94. New York, Institute of Physical Medicine and Rehabilitation, Speech and Hearing Therapy Department, 400 E. 34th St.
Auspices: New York University Bellevue Medical Center
Administrative Head: Martha L. Taylor, M.A., AS
Medical Supervisor: John Daly, M.D.

95. New York, Hearing and Speech Clinic, Manhattan Eye, Ear and Throat Hospital, 210 E. 64th St.
Auspices: Manhattan Eye, Ear and Throat Hospital
Administrative Head: Mary Wood Whitehurst, M.A., M
Medical Supervisor: Richard J. Bellucci, M.D.

96. New York, Department of Audiology and Hearing Research Center, New York Eye and Ear Infirmary, 218 Second Ave.
Auspices: New York Eye and Ear Infirmary
Administrative Head: J. A. DePew Jr.
Medical Supervisor: J. Swift Hanley, M.D.

97. New York, Speech and Hearing Center, 525 E. 68th St.
Auspices: New York Hospital, Cornell Medical Center
Administrative Head: James A. Moore, M.D.
Medical Supervisor: James A. Moore, M.D.

98. New York, New York League for the Hard of Hearing, 480 Lexington Ave. N.
Auspices: Board of Directors
Administrative Head: Eleanor C. Ronnei, M.A., AHP
Medical Supervisor: Edmund P. Fowler Sr., M.D.

99. New York, Queens College Speech and Hearing Clinic, Flushing, Queens
Auspices: Queens College
Administrative Head: Jon Eisenson, Ph.D., AS
Medical Supervisor: Medical Consultant Panel, including Otto Risch, M.D.

100. New York, Hearing and Speech Department, Amsterdam Ave. and 113th St.
Auspices: St. Luke's Hospital

Administrative Head: Erik Stenersen,
Medical Supervisor: James C. Kirkbright, M.D., F.A.C.S.

101. Rochester, Rochester Hearing and Speech Society, Inc., 800 Main St. E.
Auspices: Rochester Community Chest
Administrative Head: Irwin Brown, Ph.D., AS
Medical Supervisor: C. Stewart Nash, M.D.

102. Syracuse, Gordon D. Hoople Hearing and Speech Center, 805 S. Crouse Ave.
Auspices: Syracuse University
Administrative Head: Louis M. DiCarlo, Ed.D., F
Medical Supervisor: Gordon D. Hoople, M.D.

* 103. Utica, Hearing and Speech Center, Children's Hospital Home, 1675 Bennett St.
Auspices: Children's Hospital Home, New York State Association for Crippled Children
Administrative Head: Karl Gruppe, M.D.
Medical Supervisor: Karl Gruppe, M.D.

NORTH CAROLINA

104. Winston-Salem, Hearing and Speech Center, North Carolina Baptist Hospitals, S. Hawthorne Rd.
Auspices: Bowman Gray School of Medicine, Department of Otolaryngology
Administrative Head: Malcolm B. McCoy, Ph.D., AHP
Medical Supervisor: James A. Harrill, M.D.

OHIO

105. Athens, Ohio University Speech and Hearing Clinic
Auspices: Ohio University
Administrative Head: A. C. LaFollette, Ph.D., AS
Medical Supervisor: Francis Schmidt, M.D.

106. Bowling Green, Speech and Hearing Clinic, Bowling Green State University
Auspices: Bowling Green State University
Administrative Head: Melvin Hyman, Ph.D., AS
Medical Supervisor: None

107. Cincinnati, Cincinnati Speech and Hearing Center, 3006 Vernon Pl.
Auspices: Community Chest and Council of the Cincinnati Area
Administrative Head: Warren E. Johnson, Director, M.A., BH
Medical Supervisors: Samuel Seltz, M.D.; William H. Craddock, M.D.

108. Cleveland, The Cleveland Hearing and Speech Center, 11206 Euclid Ave.
Auspices: Cleveland Welfare Federation and Western Reserve University
Administrative Head: George J. Fortune, Director, M.A., APA, APHA
Medical Supervisor: Medical Advisory Subcommittee of Otologists

109. Columbus, Columbus Hearing Society, 209 S. High St.
Auspices: Private organization
Administrative Head: Samuel M. Cohen, Ed.M., BS
Medical Supervisor: None

110. Columbus, Ohio Department of Health, Division of Child Hygiene
Auspices: Ohio Department of Health
Administrative Head: William A. Grimm, M.A.
Medical Supervisor: Margot D. Hartmann, M.D.

111. Columbus, Speech and Hearing Clinic, Ohio State University
Auspices: Speech Department, Ohio State University
Administrative Head: Henry M. Moser, Ph.D., AS
Medical Supervisor: William H. Saunders, M.D.

112. Kent, Kent State University Speech and Hearing Clinic
Auspices: Kent State University
Administrative Head: John R. Montgomery, Director, M.A., AS
Medical Supervisor: Richard Stahl, M.D., Consultant

113. Toledo, Toledo Hearing League, 2313 Ashland Ave.
Auspices: Red Feather Agency
Administrative Head: Mrs. George Reisbach
Medical Supervisor: Nine otologists on advisory board

114. Youngstown, Youngstown Hearing and Speech Center, 69 Illinois Ave.
Auspices: Voluntary Agency; Community Chest Support; American Hearing Society
Administrative Head: May Vetterle, M.A., BH
Medical Supervisor: Committee of otologists

OKLAHOMA

*115. Oklahoma City, Oklahoma University Speech and Hearing Clinic, Norman, Okla., and Crippled Children's Hospital, Oklahoma City
Auspices: University of Oklahoma
Administrative Head: John W. Keys, Ph.D., AH
Medical Supervisors: O. A. Watson, M.D.; Chester McHenry, M.D.

OREGON

116. Portland, Portland Center for Hearing and Speech, 402 Education Center Building, 220 SW Alder
Auspices: Private social agency and United Fund
Administrative Head: George Leshin, M.Ed.
Medical Supervisor: David D. DeWeese, M.D.

PENNSYLVANIA

117. Harrisburg, Speech and Hearing Rehabilitation Centers, Room 3, Kline Village
Auspices: Department of Public Instruction
Administrative Head: Gladys B. Fish, M.Ed., BS
Medical Supervisor: None

118. Lancaster, Hearing Conservation Center of Lancaster County, 427 N. Duke St.
Auspices: Lancaster County Community Chest
Administrative Heads: Juliet C. Stephan, R.N.; Mary Alice Hunter, M.A., Advisor
Medical Supervisor: Lloyd S. Hutchison, M.D.

*119. Philadelphia, Hospital of the University of Pennsylvania, 3400 Spruce St.
Auspices: University of Pennsylvania
Administrative Head: Robert J. Hunter, M.D.
Medical Supervisor: Robert J. Hunter, M.D.

120. Philadelphia, St. Christopher's Hosiptal Speech and Hearing Center, Lawrence and Huntingdon Sts.
Auspices: EENT Department, St. Christopher's Hospital for Children
Administrative Head: David Myers, M.D.
Medical Supervisor: A. Neil Lemon, M.D.

121. Philadelphia, Speech and Hearing Rehabilitation Centers, St. Christopher's Hospital for Children, Lawrence and Huntingdon Sts.
Auspices: Department of Public Instruction
Administrative Head: Milton N. Brutten, Ph.D.
Medical Supervisor: None

122. Philadelphia, Temple University Medical Center, 3401 N. Broad St.
Auspices: Department of Otology, Temple University School of Medicine
Administrative Head: Philip Rosenberg, Ph.D., BH, AHP
Medical Supervisor: David Myers, M.D.

123. Philadelphia, U. S. Naval Hospital, National Naval Aural Rehabilitation Center, 17th and Pattison Ave.
Auspices: U. S. Navy
Administrative Head: Joseph A. Scanlon, A. B., Coordinator of Services
Medical Supervisor: Captain William C. Livingood (MC), USN.

124. Pittsburgh, Pittsburgh Hearing Society, 313 Sixth Ave.
Auspices:
Administrative Head: Phyllis McCalmont, M.A.
Medical Supervisor: Professional advisory committee

125. Pittsburgh, The Speech and Hearing Rehabilitation Center, Eye and Ear Hospital, 230 Lothrop St.
Auspices: Department of Public Instruction
Administrative Head: Elmer Owens, Audiologist, M.A.
Medical Supervisor: None

126. Pittsburgh, Department of Audiology, School of Medicine, 230 Lothrop St.
Auspices: Eye and Ear Hospital and University of Pittsburgh School of Medicine
Administrative Head: Leo G. Doerfler, Ph.D., AH, F
Medical Supervisor: Kenneth M. Day, M.D.

127. Reading, Clinic for the Hard of Hearing, Reading Hospital
Auspices: Reading Hospital
Administrative Head: James E. Landis, M.D.
Medical Supervisor: James E. Landis, M.D.

128. Scranton, The Speech and Hearing Rehabilitation Center, 1800 Washington Ave., The Pennsylvania State Oral School for the Deaf
Auspices: Department of Public Instruction
Administrative Head: Albert F. Johnson, Audiologist, M.A., BH
Medical Supervisor: None

129. University Park, The Speech and Hearing Rehabilitation Center, Pennsylvania State University
Auspices: Department of Public Instruction
Administrative Head: Raymond J. Lczak, Audiologist, M.A., M
Medical Supervisor: None

130. University Park, Speech and Hearing Clinic, Pennsylvania State University
Auspices: Pennsylvania State University
Administrative Head: E. T. McDonald, D.Ed., AS
Medical Supervisor: Tom Mebane, M.D., Consultant

SOUTH CAROLINA

131. Greenville, Greenville Hearing and Speech Center, Greenville General Hospital
Auspices: United Fund of Greenville County
Administrative Head: Mervyn L. Falk, M.A., BSP
Medical Supervisors: A. J. Goforth, M.D.; Paul P. Hearn, M.D.

TENNESSEE

132. Chattanooga, Speech and Hearing Center of Chattanooga-Hamilton County, 605 Vine St.
Auspices: Nonprofit corporation
Administrative Head: J. Dale Welsch, Ed.D., AS
Medical Supervisor: Harold Alper, M.D.

133. Memphis, Memphis Speech and Hearing Center, 874 Monroe St.
Auspices: Easter Seal Society; University of Tennessee College of Medicine; Memphis Junior League; East Memphis Exchange Club; Council of Jewish Women
Administrative Head: John A. Irwin, M.A., AHP
Medical Supervisor: Sam Sanders, M.D.

134. Nashville, The Bill Wilkerson Hearing and Speech Center, 2109 Garland Ave.; or Vanderbilt University Hospital
Auspices: Nonprofit corporation
Administrative Head: Freeman McConnell, Ph.D., AH
Medical Supervisor: W. W. Wilkerson Jr., M.D.

TEXAS

135. Austin, University of Texas Speech and Hearing Clinic, Speech Building
Auspices: Department of Speech, University of Texas
Administrative Head: Jesse J. Villarreal, Ph.D., AS, Director
Medical Supervisor: Douglas F. Barkley, M.D.

136. Dallas, Audiology and Speech Correction Clinic, Veterans Administration Hospital, 4500 S. Lancaster Rd.
Auspices: Veterans Administration
Administrative Head: Burton B. King, Acting Chief, M.A., M
Medical Supervisor: Ludwig A. Michael, M.D.

137. Galveston, Hearing and Speech Clinic, The University of Texas Medical Branch, 105 Kieller Building
Auspices: The University of Texas Medical Branch
Administrative Head: C. Olaf Haug, M.A., AH
Medical Supervisor: Fred R. Guilford, M.D.

138. Houston, Houston Speech and Hearing Center, 6504 Bertner
Auspices: United Fund and American Hearing Society
Administrative Head: Jack L. Bangs, Ph.D., AS
Medical Supervisor: Diagnosis and Research Committee from Board of Directors

UTAH

139. Salt Lake City, Speech and Hearing Center, 1699 E. Fifth South (Hempstead Rd.), The University of Utah
Auspices: The University of Utah
Administrative Head: Wallace A. Goates, Ph.D., AH
Medical Supervisor: David A. Dolowitz, M.D.

VERMONT

140. Rutland, Children's Rehabilitation Center, 88 Park St.
Auspices: Vermont Association for the Crippled, Inc.
Administrative Head: Miss Harriet M. Dunn, M.A., AS, Director of Speech and Hearing Services
Medical Supervisor: Ralph H. Seeley Jr., M.D.

VIRGINIA

141. Charlottesville, Speech and Hearing Center, 109 Cabell Hall, University of Virginia
Auspices: University of Virginia
Administrative Head: James M. Mullendore, Ph.D., AS
Medical Supervisor: Fletcher D. Woodward, M.D.

142. Richmond, Hearing and Speech Center, Medical College of Virginia, Box 846, 231 N. 12th St.
Auspices: Medical College of Virginia
Administrative Heads: S. James Cutler (Hearing), B.S.; John Collison (Speech), M.A., BS
Medical Supervisor: P. N. Pastore, M.D.

WASHINGTON

143. Seattle, Seattle Hearing and Speech Center, 1229 10th Ave. N.
Auspices: King County Community Chest
Administrative Head: Frederick J. Artz, B.A., BH
Medical Supervisor: John T. Tolan, M.D.

144. Spokane, Inland Speech and Hearing Clinic, 304 Paulsen Building
Auspices: Private
Administrative Head: John W. Veatch, B.A., BS, BHP, ASP
Medical Supervisor: None

WISCONSIN

145. Milwaukee, Hearing Evaluation Center, University of Wisconsin, 3203 N. Downer Ave.
Auspices: University of Wisconsin
Administrative Heads: R. Myers, Chairman, Speech Department, M.A.; R. Briskey, Acting Director, M.A., BS
Medical Supervisor: Meyer Fox, M.D.

University of Denver, University Park (10).

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Case Reports

A Mass in the Neck

D. S. De STIO, M.D., Pittsburgh

A mass in the neck frequently signifies the presence of carcinoma of the nasopharynx. The nasopharynx should be carefully examined, not only by means of the mirror but also by digital palpation and biopsy when a neck mass is suspected of being a metastatic malignancy.

It is better to completely excise a small neck mass for diagnosis rather than perform a needle biopsy unless the mass is so large that its complete resection is improbable. There is always some danger of not getting representative tissue in the needle biopsy and thereby arriving at an incorrect diagnosis. If the primary carcinoma is discovered in the nasopharynx, irradiation of the primary and metastatic area in the neck would be the treatment of choice.

Report of Case

A 60-year-old white man was first examined in my office on Jan. 9, 1956. His only complaint was a stuffiness in both sides of his nose and gradual equal bilateral deafness. Both complaints were of one year's duration. Examination showed that the mouth, oral pharynx, and larynx were normal. The nasopharynx was clearly and easily seen with a mirror and appeared normal. The nose and sinuses were normal except for a moderate hypertrophy of both inferior turbinates. The ear drums were normal. The Rinne test was positive, and the audiogram showed an early bilateral nerve-type deafness, equal in both ears. Nasal secretions did not show evidence of allergy. Diagnosis of hypertrophic turbinates was made, and submucous electrocoagulation of both lower turbinates was advised.

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Associate Professor of Rhinolaryngology, University of Pittsburgh.

Read before the Section on Laryngology, Otology and Rhinology at the 105th Annual Meeting of the American Medical Association, Chicago, June 12, 1956.

While the neck was being examined, a small, painless, firm but not hard, round, smooth, freely movable mass about 1½ cm. in diameter was palpated but hardly visible just below the angle of the left mandible. This was thought to be an enlarged lymph node, and, upon questioning the patient about it, he said that it had been there about one month and that, since it did not disturb him in any way, he had not paid much attention to it.

Electrocoagulation of the right lower turbinate was done. One week later, on Jan. 16, the left lower turbinate was electrocoagulated, at which time I noted a paralysis of the external rectus muscle of the right eye. The patient stated that he had noted it a few hours after the right electrocoagulation treatment one week previously.

Complete blood count, Kahn test, urinalysis, x-rays of skull, neck, and chest were all normal. The ophthalmologist confirmed the ocular paralysis but could find no cause.

The patient failed to return to my office for about two months, reporting on March 10, 1956, at which time he said that the nasal stuffiness had completely disappeared but that some numbness and pain had developed over the right cheek and that he was worried about his paralyzed right eye.

The small mass in the left side of neck had not changed in size. Mirror examination of the nasopharynx showed no abnormality. I became so strongly suspicious of nasopharyngeal carcinoma that I had the patient admitted to the hospital on March 16 and performed a biopsy of the right side of the posterior nasopharyngeal wall under local anesthesia. At the same time I digitally palpated the nasopharynx and noted very slight, hard, smooth forward bulging of the entire posterior nasopharyngeal wall, which was not evident by direct or mirror examination. X-ray of head, neck, and chest were again all negative.

The pathologist was unable to make a diagnosis and reported that the tissue was probably malignant but that a granulomatous lesion could not be excluded. On March 25 biopsy was again taken from both sides of the nasopharynx and the entire neck mass was excised. Microscopic report was primary transitional-cell carcinoma of

all nasopharyngeal specimens with metastasis to the cervical lymph node.

Deep x-ray treatments to the nasopharynx and neck were started immediately, the patient receiving 35 of them for a total of 12,525, r, until they were discontinued on May 11, 1956.

Comment

1. Careful study of the nasopharynx is always indicated when a mass in the neck is present.

2. Digital palpation of the nasopharynx may be more helpful than mirror examination in certain cases of nasopharyngeal carcinoma.

Summary

A mass in the neck often presents difficult problems in diagnosis. It may represent the first demonstrable evidence of serious disease of the upper air or food passages. Careful nasopharyngeal inspection and palpation, including blood, x-ray, and biopsy studies, should be promptly made so that appropriate treatment may be instituted.

121 South Highland Avenue.

Nail Removed from Duodenum with Intraluminal and Extracorporeal Magnets

MURDOCK EQUEN, M.D.; GEORGE ROACH, M.D.; ROBERT BROWN, M.D., and TRUETT BENNETT, M.D., Atlanta

From the dawn of history magnetism has fascinated the minds of men and many efforts have been made to apply its properties in scientific medicine. Charlatans have found magnetism a fertile field: In cheap magazines one may still see advertisements of "magnetic belts" and the like, good for whatever ails you. It has long been established, however, that the usefulness of the magnet is limited to the removal from the body of objects that respond to its pull.

The electromagnet has often served to extract spicules of steel from the eye, but with modern safety precautions it is seldom needed now. Ten years after World War I, Jackson[1] demonstrated a steel-jacketed machine gun bullet high in an abscess cavity in the upper lobe of the left lung of a Canadian veteran. Unable to reach the bullet with forceps, he employed an electromagnet over the rib margin in front to draw it down to the lower part of the cavity. There he picked it up with forceps and slowly withdrew the bullet through a strictured fistula. The use of electromagnets externally as an adjunct in bronchoscopy has not been common since then.

The General Electric Company in 1943 perfected a powerful, permanent magnet of an alloy of iron, aluminum, nickel, and cobalt, which they called Alnico. One of us realized the possibilities of a magnet small enough to be swallowed or to insert through a bronchoscope, and reported success with it at the meeting of the Southern Medical Association in St. Louis, November, 1944.[2] Many subsequent communications have issued from the Ponce de Leon Infirmary. In these papers have been described various modifications in the size and shape of the magnet as well as refinements in its application. A straight magnet has been fixed to the end of a semiflexible stylet (such as a ureteral catheter) for recovering foreign bodies in the smaller bronchi; this type is also advocated for pushing an open safety pin into the dilated stomach, there reversing it before pulling it back up the esophagus. A curved magnet has been devised, which, attached to a string, can be swallowed and retrieved after contact with the foreign body. Then the string was made radiopaque to be sure that it does not get tangled up. This plastic-coated string was developed by the School of Physics at the Georgia Institute of Technology. More recently a cap has been fitted over the proximal end of the magnet through which the string passes in order that the pull may be exerted in a straight line. It has been emphasized repeatedly that the preoperative use of a Berman locator to determine whether the foreign body will respond to the magnet is important.

In 1947 we saw a little boy who had harbored a swallowed nail in his duodenum so long that its further progress could not be expected. We had him swallow a magnet attached to a string and checked him with the fluoroscope until the magnet and nail came together. With constant fluoroscopic control, we then pulled back magnet and

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From the Ponce de Leon Eye, Ear, Nose and Throat Infirmary.

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nail. The operation took less than two minutes. At the meeting of the American Medical Association in Atlantic City, N. J., that June we reported the case, claiming that it was the first time that a foreign body had been removed from the duodenum via the stomach and esophagus.[3] This claim has not been challenged, not even by the Russians.

Since this first case we have seen 23 children with ferrous bodies in the duodenum. In two the magnet dislodged the nail, but the nail slipped away and eventually was passed through the rectum, not without causing considerable concern meanwhile to the family and to us. We have succeeded in recovering 8 more nails and 12 bobby pins from the duodenum with the magnet. In one case, however, even three Equen magnets were not strong enough to get the nail and it was necessary to resort to open operation.*

To avoid the dangers of major surgery and longer hospitalization when a small magnet in contact with a nail or bobby pin in the duodenum was not enough to bring it out, whether held by inflammatory reaction or folds of the intestine, we reasoned that larger magnets outside of the body might bring success.

Report of Case

An infant of 15 months swallowed a six-penny nail in another city. An x-ray on Sept. 8, 1954, showed the nail lying free in the stomach. A week later a second x-ray proved that it had passed into the first portion of the duodenum, and on the 23rd its position was unchanged. He was referred to us on Sept. 29.

Anteroposterior and lateral films at the Ponce de Leon Infirmary on admission revealed the nail in the right midquadrant of the abdomen, near the back, with the point up. It was diagnosed as being in the descending limb of the duodenum with the point just beyond the pylorus. On comparing our plates of the 29th with those taken elsewhere on the 15th and the 23rd, it was apparent that the nail had not made any progress since the 15th, and it was considered impossible that it could be carried beyond the ligament of Treitz by normal peristalsis.

Fig. 1.—The six-penny nail is shown in the first portion of the duodenum, point up. The Equen curved magnet attached to a radiopaque string is in contact with the point of the nail.

A curved Equen magnet attached to a radiopaque string was introduced into the baby's pharynx and pushed by the surgeon's finger into the esophagus. The child's arms were put in splints to prevent his snatching the string out. Within four hours the fluoroscope showed that the magnet had established contact with the nail in the duodenum. Traction on the string failed to budge the nail.

A bar magnet of Alnico, weighing about 8 lb.,† was placed on each side of the infant's body so that the magnetic field would pass through the nail. These magnets outside the body exerted an appreciable torque on the nail and induced magnetism in it so that there was a stronger force of attraction between the nail and the intraduo-

* In this case Dr. Harry Rogers opened the abdomen and removed the nail, which had perforated the third portion of the duodenum, and the three magnets, two of which were lying free in the peritoneal cavity. Convalescence was uneventful, but it took more than three days for the child to recover from such an operation.

† These magnets were loaned to us by Prof. Joseph H. Howey, Director, School of Physics, Georgia Institute of Technology.

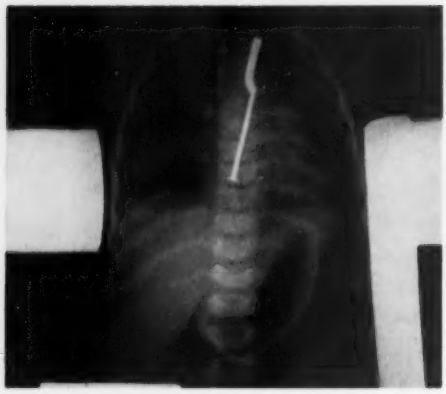

Fig. 2.—The magnet and the nail are being drawn back up the esophagus. The large Alnico magnets may still be seen at each side of the body.

denal magnet. Again, constant moderate traction was exerted on the string. The positions of the large magnets were changed several times when suddenly the nail was seen to break free. Still attached to the Equen magnet, it was quickly withdrawn through the stomach into the esophagus, always under fluoroscopic guidance. At the cricopharyngeal constriction the nail was stripped from the magnet, but with the help of a small esophagoscope and forceps it was promptly brought out.

After being kept under observation three days, the child was allowed to go home, as well as ever.

Summary

Presented is the case of a 15-month-old child who had harbored a six-penny nail in the first portion of the duodenum something more than two weeks. Since this nail could not be budged with an Equen magnet alone, 8-lb. Alnico magnets were employed outside the body to dislodge it, and the nail and small magnet were then quickly withdrawn through the stomach and esophagus.

144 Ponce de Leon Ave., N. E. (8).

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Progress in Otolaryngology

Bronchoesophagology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1955

F. JOHNSON PUTNEY, M.D., Philadelphia

Bronchoscopy was utilized to a great extent in performing cardioangiography during 1955, with satisfactory results and no serious complications. Cytologic study of bronchial secretions retained its important diagnostic position in bronchogenic carcinoma. While some reports appeared in the otolaryngological literature on the surgical correction of pulsion pharyngoesophageal diverticulum, the chief interest in the esophagus centered upon the lower end, with esophagitis again claiming a major share of the discussion.

Experimental Studies

Experimental transbronchial cardioangiography was performed by Taufic and Asta.[1] A bronchoscope was inserted into the left main bronchus just distal to the carina, and a 15 gauge needle fitted into a saline-filled aspirating tube was inserted into the left atrium through the wall of the left main stem bronchus at a point 1 cm. from the carina. Aspiration of bright red blood into the syringe confirmed the presence of the tip of the needle in the atrium, and Diodrast (3,5-diiodo-4-pyridone-N-acetic acid diethanolamine) was injected. Effective cardioangiographic detail of the left side of the heart, interventricular septal defects, and the aorta was obtained by this method.

The effect of Dionosil (aqueous suspension of the *n*-propyl ester of 3,5-diiodo-4-pyridone-N-acetic acid with the addition of sodium carboxymethylcellulose) and Dionosil Oily in the bronchi of dogs was studied by Joynt and Harnick.[2] The material produced a coating on the bronchial wall which clearly outlined the bronchi with minimal alveolar spread. No granulomatous reaction was noted in the lungs at the end of four weeks after use of the aqueous solution.

Experimental studies on animals with the use of aqueous and oily media in the lungs were carried out by Smith and Harrill,[3] who used the water-soluble preparation iodopyracet injection (Umbradil Viscous B) and the oily preparation chloriodized oil (Iodochlorol). Saline was used as a control. The lungs of all the animals developed an acute patchy bronchopneumonia in intervals up to 10 days and the reaction persisted throughout this time with both the oily and aqueous media, while the animals treated with saline showed a marked reaction in 48 hours but marked clearing in 6 days.

The tolerance of the bronchial wall to high-level dosage of radioactive colloidal gold following experimental intrabronchial injection was studied by Berg and his coworkers.[4] The radiation effect was confined to the injection site and its lymph drainage area. No untoward effects at the site of injection with the use of relatively high doses were noted, indicating that mobilization from the mucosa to the lymph nodes takes place rapidly before severe local injury ensues. This method of delivering radiation was suggested for palliation of patients with inoperable bronchogenic carcinoma.

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Physiology

Batson [5] confirmed the clinical observations on the cricopharyngeus muscle and established its definite anatomic-physiologic structure. The horizontal bundle of fibers was found to be a true sphincter of the esophagus, and various degrees of tonicity occurred.

Resting and deglutition pressures in the pharynx and upper portion of the esophagus were recorded by Fyke Jr. and Code.[6] The findings indicated that the upper part of the esophagus was closed off from the pharynx under resting conditions by tonic contraction of the cricopharyngeus sphincter, confirming the belief that its "pinch-cock" effect is a true sphincter. The sphincter was also found to contract on deglutition and was coordinated with the peristaltic contraction of the pharynx above and the esophagus below. The biphasic nature of the pressure changes in this region with both positive and negative components was established. An orderly progression of the high pressure wave through the pharynx to the esophagus was noted, indicating that coordinated peristaltic contraction occurs during deglutition.

Peters [7] discussed the physiology of the lower end of the esophagus with special reference to the closure mechanisms at the cardia. Three major closure mechanisms were recognized: (1) the extrinsic diaphragmatic hiatal sphincter (pinch-cock) working in conjunction with the elastic diaphragmaticoesophageal ligament, (2) the intrinsic cardiac sphincter action, and (3) the valvular mechanism. It was concluded that each of these three major closure mechanisms operated to some degree and that the system as a whole was an intricate and complex one. It was difficult for the author to subscribe to the feeling that a particular mechanism played no part in a lesion because its impairment did not lead to regurgitation, for adaptation of those remaining mechanisms may be sufficient to maintain comparative efficiency.

A comparison of experimental esophageal pain with the clinical pain of angina pectoris and esophageal disease was studied by Kramer and Hollander.[8] Variation in the location and type of pain produced by esophageal balloon distention in 29 persons with normal esophagi was considerable. Distention of the upper esophagus produced pain in the area of the balloon, but distention of the middle and lower thirds resulted in scattering of the pain, generally referred below the balloon level. The commonest finding was some type of pressure pain. Seven of nineteen patients with angina pectoris could not distinguish the angina pain from the esophageal distention pain, and no EKG changes occurred during esophageal distention. In 4 of 10 patients with esophageal disease balloon distention did not reproduce the clinical pain of the disease.

The pain produced by distention of a balloon in the lower esophagus was studied in eight normal subjects and in three subjects with coronary artery disease by Boylis and his associates.[9] Esophageal pain differed from the pain of cardiac ischemia in character, fluctuation, speed of precipitation and abolition, and absence of electrocardiographic changes. The only cardiographic change noted was in achalasia when an axis shift alone occurred.

Bronchology

Delivering the Semon Lecture, Huizinga [10] discussed the science of bronchological pneumonology in its broad and basic aspects. The new term bronchological pneumonology was used to emphasize the fact that pulmonary abnormalities are often the result of bronchial disease, usually bronchial stenosis. As the name indicates, bronchoscopy should always be considered in pulmonary affections, and the science has now become the borderline between general medicine and laryngology.

Holinger [11] defined the role of the laryngologist in resuscitation of the newborn infant as one of establishing and maintaining an airway. In the examination of a

cyanotic and dyspneic infant the lesion must first be localized, and if it proves to be respiratory in origin it is necessary to further investigate the airway for obstruction. In the event that the airway is obstructed it must then be determined whether the obstruction is nasopharyngeal, laryngeal, or tracheobronchial. Direct laryngoscopy and bronchoscopy are essential, and tracheotomy may also be necessary when pathologic changes responsible for the obstruction are found and can be relieved by no other methods. The laryngologist is frequently called upon to aspirate obstructing secretions in newborn infants; but if the obstruction is severe enough or the secretions copious enough to necessitate this procedure, usually some underlying congenital anomaly or birth injury is found as the basic pathologic responsible condition and the secretions themselves are secondary to this change.

In discussing bronchoscopy in the diagnosis and treatment of bronchopulmonary disease Putney [12] considered this method valuable in obscure pulmonary disease, and in determining the cause of unexplained pulmonary symptoms and the etiology of bronchial obstruction, both complete and partial. It also proved useful in the treatment of bronchial obstruction when due to foreign bodies, benign tumors, retained secretions, or fibrostenosis. Bronchoscopy was the best means of providing a positive diagnosis in bronchogenic carcinoma both by histologic and cytologic study of tissue and secretions and in determining preoperatively the limits of the growth to ascertain its operability.

Electrocardiographic studies during bronchoscopy on patients with significant cardiovascular disease were performed by Maloney and Massey.[13] In a total of 32 bronchoscopies there was no change in the electrocardiogram in 25. One had inconsequential changes, and six had significant changes. Although there were no deaths during bronchoscopy, two deaths occurred within a few hours following bronchoscopy (attributed to the procedure or the premedication). Seven additional patients died later, but the bronchoscopy was not a factor in their death. It was concluded that the risk of bronchoscopy in patients with cardiovascular pathologic lesions was overstressed and that conventional indications for bronchoscopy were also applicable to these patients. Special caution was needed in cases with major arrhythmias, for these variations were likely to become significant upon instrumentation.

Cardioangiographic Pressure Measurements

Allison and Linden [14] further extended the measurement of pressures in the left auricle through the bronchoscope to measuring pressures in the pulmonary artery and aorta. This procedure was employed in 121 patients without death or disability and proved no more uncomfortable to the patient than simple bronchoscopy provided the local anesthesia was thorough and effective and the technical procedure was performed smoothly. Puncture of the pulmonary artery was performed through the anterior wall of the trachea just above the carina, while the aorta was reached by inserting the needle 2-3 cm. higher in the trachea. Typical pressure-pulse curves from these vessels were described and have been valuable in assessing the status of the mitral valve.

Pulmonary Pressure Measurements

A comparison of esophageal and intrapleural pressures in 14 patients was made by Cherniack et al.[15] using a thin-walled rubber balloon in both conscious and anesthetized subjects. The relationship between the esophageal and intrapleural pressures was found to be inconsistent and variable. The pulmonary elastic pressure measured by the intrapleural pressure was always greater than that found by using esophageal pressures. The nonelastic pulmonary pressures by the two methods were approximately the same. Because of the extreme variation encountered the relative merits of

intrapleural and esophageal pressure measurements were inconclusive.

Anesthesia

Gierson and his co-workers [16] found alphaprodine (Nisentil), which was used in 119 bronchoscopies, an acceptable analgesic agent for bronchoscopy in 96.6%. The drug's induction time was 10 to 15 minutes, with a duration of approximately 2 hours. It did not interfere with the patient's ability to cooperate, and the side-effects were limited to mild transient dizziness with no after-effects. The best results were obtained with 30 mg. injected subcutaneously preceded by sodium pentobarbital, 1½ grains (0.1 gm.), orally.

In a panel discussion on anesthesia in bronchoesophagology Roberts [17] was of the opinion that the only justification for not using anesthesia in children was respiratory distress. Relaxant drugs such as curare in divided doses administered by intravenous infusion followed by thiopental (Pentothal) sodium are gaining favor among bronchoesophagologists. Mutual understanding between the surgeon and the anesthetist minimizes the problems of anesthesia, and vigilant and constant supervision of the patient is necessary, for there always remains some hazard. The following requirements are necessary for safe and adequate anesthesia: The patient must have no pain and be sufficiently relaxed to permit adequate observation, and the anesthetic agent must be safe. No anesthetic agent or technique is applicable or desirable in all patients and must be selective and individualized.

Holinger [18] used for the majority of peroral endoscopic examinations a local anesthesia consisting of spraying the larynx with 2% tetracaine (Pontocaine) and instilling 2 cc. of 10% cocaine into the trachea and larynx with a laryngeal syringe under mirror guidance. Three to five minutes was allowed to elapse between the spraying of the tetracaine and the laryngeal instillation of cocaine, and it was brought out that toxicity was based not only on total dosage but also on rate of absorption. While preoperative medication depended on the age and general condition of the patient, in routine cases adults were given 50 mg. of meperidine (Demerol) and 2 grains (0.12 gm.) of sodium phenobarbital hypodermically approximately one hour preoperatively. Atropine was used in addition for laryngoscopy and esophagoscopy but omitted for bronchoscopy. In old or debilitated persons or in those with respiratory obstruction no premedication was given. As a rule no local or general anesthesia was used in infants or children, and sedation using morphine or meperidine and a barbiturate was used unless respiratory obstruction was present. Many variables were found in evaluating the effectiveness of a local anesthetic.

Jackson [19] emphasized the importance of technique of administration of local anesthesia and concluded that the majority of work can be done under local anesthesia or, in the case of infants and young children, without anesthesia. His technique consisted of first using a pharyngeal spray of 2% tetracaine and then fractionated instillation of 10% cocaine solution into the tracheobronchial tree under the guidance of a laryngeal mirror. One death due to tetracaine used for a bronchogram and one death due to cocaine with other mild reactions occurred. Premedication consisted of pentobarbital (Nembutal), meperidine, and atropine. Local or topical anesthesia was considered safer, particularly when working in the dark under fluoroscopic guidance, and in the presence of a large foreign body or lesions in the esophagus. The safety factor was increased and simplified for the endoscopist and nurses as well as the patient if the procedure was done smoothly and satisfactorily under local anesthesia. Psychological preparation of the patient along with local anesthesia played a part, and in endoscopy in the child proper control of the patient and holding of the head were also important.

General anesthesia for endoscopic examinations was discussed by Stage,[20] who con-

cluded that although generally speaking topical anesthesia was less hazardous than general anesthesia in this particular field, general anesthesia was indicated in certain conditions. The importance of careful preoperative and postoperative evaluation and medication was stressed, and neither the surgeon nor the anesthetist should consider it a minor matter. A few problems in general anesthesia were as follows: the asthmatic patient; laryngeal edema and the endotracheal tube; granuloma and the endotracheal tube; preoperative cortisone and corticotropin (ACTH); postoperative use of cortisone; the dangers of chlorpromazine (Thorazine); cardiac arrest and fibrillation; nasal, oral, and tracheal reflexes; convulsions; the curares and prolonged apnea; laryngeal and bronchial spasms; patients with bleeding problems.

Shane and Ashman [21] described a method of general anesthesia for bronchoscopy and bronchography using a combination of intravenous and inhalation anesthesia accompanied by the injection of a local anesthetic solution (tetracaine) through the cricothyroid membrane. The advantages claimed for this method of anesthesia were safety, consistence of plane of anesthesia, absence of ether, lengthening of the time of the procedure without apprehension on the part of the patient or speed on the part of the surgeon, recovery from anesthesia within three to seven minutes after its termination, elimination of the danger of toxic reaction from local analgesia, and usefulness in all age groups, including children.

A technique for endoscopic procedures using induced paralysis obtained by succinylcholine chloride intravenously unaccompanied by either topical or general anesthesia was used in 22 cases by Alver and Leek.[22] The advantages claimed were those of complete relaxation of the patient, making the technical introduction of the instrument easier; absence of gagging or coughing; ready accessibility of the bronchial divisions to direct vision; absence of reactions from topical anesthetic agents, and comfort of the patient.

Palmer and Deutsch [23] experienced 15 instances of reaction to local anesthesia with one sudden death during preparation for esophagoscopy with a gargle of tetracaine. The fatality was considered due to tetracaine sensitivity.

Bronchoscopic Technique

The elder Jackson [24] pointed out that the laryngoscope was not necessary for the introduction of the bronchoscope and was not used by experienced bronchoesophagologists for routine bronchoscopy. It was considered advisable to use the laryngoscope for bronchoscopic introduction in cases in which there was imminent asphyxia to secure prompt exposure of the larynx and make introduction of the bronchoscope easier and also allow visualization of the laryngeal airway. In children exposure of the larynx with a laryngoscope before introduction of the bronchoscopy not only simplified the technique but also allowed inspection of the larynx and determination of the amount of subglottic edema and laryngeal inflammation. The laryngoscope aided in obtaining laboratory specimens for research work which were not exposed to mouth secretions. In training younger men to acquire skill the laryngoscope was used initially in order to obtain familiarity with the larynx and enable them to locate it easily so that the bronchoscope could be introduced without additional trauma.

Bedside bronchoscopy was performed by Schaff [25] most frequently for the treatment of postoperative atelectasis or for bronchial aspiration of patients in coma. Aspiration was done in bed to avoid exertion, since many of the patients were anoxic and there was an elevated pulmonary arterial pressure as well as increased cardiac output. Oxygen was used only for anoxic patients, and preoperative medication was avoided. Satisfactory positioning was secured by placing the patient diagonally across the bed, and satisfactory anesthesia was obtained by spraying the pharynx or using the transtracheal route.

Bronchospirometry

Curtis and his co-workers [26] adapted the one-breath oxygen test for bilateral alveolar nitrogen curves to differential bronchospirometry to obtain the gas distribution of each lung.

Complications of Bronchoscopy

A case of selective pneumothorax without emphysema was recorded by Friedman [27] as a complication of bronchoscopy. There were no symptoms, and the pneumothorax was discovered on roentgen examination following bronchoscopy.

Postoperative Tracheobronchial Complications

By bronchoscopic examination Dark and Jewsbury [28] detected postoperative membranous tracheobronchitis due to endotracheal anesthesia in four cases. The cause was assumed to be local trauma not from overinflation of the tube but from the rotary movement within the trachea, especially upon moving the head from side to side.

When conservative efforts to encourage coughing and remove retained pulmonary secretions that were causing atelectasis failed, Janes [29] employed bronchoscopy within a few hours, particularly if the area of atelectasis was large. Bronchoscopy was frequently used in postoperative chest cases and following severe chest and head injuries but was rarely required in other postoperative conditions.

Hemoptysis

Anderson and his associates [30] pointed out that hemoptysis of benign origin is commoner than is generally supposed and results in many cases from simple bronchial erosion produced by bronchitis. Often the bleeding area was found by bronchoscopy and appropriate etiological studies instituted. Many cases of so-called idiopathic hemoptysis may be due to minor inflammations of the bronchi in areas not accessible to bronchoscopy.

Mitchell [31] classified 30 cases of hemoptysis with normal chest roentgenograms according to the bronchoscopic appearances. Twenty-four cases comprised the nonmalignant group, of which one died of secondary adenocarcinoma seven months after hemoptysis. Of these, 13 were considered normal, 4 had bronchitis, 5 revealed mucopus arising from one bronchus, 1 had a tuberculous stricture, and 1 had an inflammatory stricture of unknown etiology. The 23 survivors of this group were reexamined after an average period of 23 months, and all except 1 with bronchiectasis had no further hemoptysis. There were six patients in the malignant group, of which four had definite carcinomas and two were doubtful. Of the two with doubtful carcinomas both were alive and well 9 months and 22 months, respectively, after bronchoscopy.

Congential Lesions

A report of a case successfully operated on for incomplete separation of the larynx and the upper trachea from the esophagus in a newborn was presented by Pettersson.[32] Direct laryngoscopy disclosed that the posterior wall of the larynx and the upper part of the trachea were not united in the midline. The posterior wall of the larynx closed spontaneously on expiration, but on deep inspiration the entire posterior wall opened from the arytenoids downward, permitting a simultaneous view into the esophagus and upper part of the trachea. Following surgical correction hypopharyngoscopy and laryngoscopy were repeatedly needed for dilatation purposes until three months after operation.

Morse and Gladding [33] reported a case of bronchial obstruction in an infant due to a misplaced left pulmonary artery. Bronchoscopy revealed that the lumen of the right bronchus was very narrow with only a few air bubbles getting in or out of the right bronchus, and the anomalous vessel was found at operation to be causing the stenosis.

Sandison [34] described an anomaly of the upper respiratory tract consisting of atresia of the larynx, partial absence of the trachea, and esophagotracheal fistula.

Valle [35] was able to collect from the literature 120 cases of agenesis of the lung. The condition was found in a 72-year-old woman, indicating that it does not prevent a normal life span, although it does diminish the ability to withstand pulmonary infections.

Battersby [36] reviewed a total of 90 patients in whom the diagnosis of congenital lesion of the esophagus was made. The diagnosis was established by roentgen studies and esophageal intubation. Iodized oil was used as the diagnostic medium only when a stenosis or cardiospasm was suspected. Esophagoscopic examination was the most helpful single aid when a tracheoesophageal fistula was present.

An unusual case of high tracheoesophageal fistula with complete obstruction of the esophagus by a membranous diaphragm was presented by Schwartz and Dale.[37]

A case of congenital atresia of the esophagus with two esophagotracheal fistulae in which the esophagus was continuous though atretic between the two fistulae was described by Krediet.[38]

Three cases of congenital tracheoesophageal fistula without esophageal atresia were presented by Herweg and Ogura,[39] who described a new endoscopic technique for diagnosis. An essentially closed system using an endotracheal tube and esophagoscope with intratracheal instillation of dye and radiopaque medium was used in two infants to localize the fistula. By this method respirations were controlled and aspiration minimized. When the medium was placed in the trachea and appeared in the esophagus the presence of a fistula was confirmed, and the use of a methylene blue saline solution was found to readily pass the smallest opening. With repeated injections of the dye the esophagoscope was moved to the fistula, and lateral roentgenograms accurately localized the level in reference to the tracheobronchial tree.

Colt and his associates [40] reported a case of congenital esophageal fistula without esophageal atresia and pointed out that this type of anomaly gives the best prognosis and is the most easily repaired surgically.

Another case of congenital tracheoesophageal fistula without esophageal atresia was reported by Pryles and Huvos,[41] who pointed out that endoscopic examination was the most exact method of determining the level of the fistula and should be performed in every case in which the location was doubtful.

Tracheal Stenosis

Tracheal stenosis occurred in six tracheotomized poliomyelitis patients (one adult and five children), and the surgical repair was described by Falbe-Hansen.[42] After excision of the scar tissue a polyethylene tube of suitable width and length was inserted and fixed through the skin with silver wire sutures. The indwelling tube was removed by direct laryngoscopy at the end of three to five months, and no further treatment was needed in five of the six cases. The polyethylene tube remained unchanged, and on removal the tracheal mucosa was grossly normal. Granulations were not observed in any case.

A case of traumatic tracheal atresia was reported by Hanckel [43] in which the injury followed lacerations of the neck from slivers of glass. Several operative procedures were performed, and finally the difficulty was overcome by secondary laryngofissure operation with longitudinal incisions in the scar tissue and insertion of an indwelling vinyl plastic intratracheal tube.

A case of severe tuberculous stenosis of the trachea requiring surgical correction was recorded by Nicks and Watt.[44]

Tracheal and Bronchial Grafts

Abbott and his associates [45] discussed the indications for tracheal or bronchial reconstruction by grafting procedures and reported successful employment of grafts in the management of acute trauma, various

types of bronchial stenosis, and neoplastic lesions. The repair of congenital defects may offer an additional field for this type of surgery, for the grafts were well tolerated in childhood and appeared to grow in a satisfactory manner with the child. Successful grafts have been used in the management of bronchogenic cyst of the tracheal wall and stricture secondary to prolonged sojourn of an endobronchial foreign body. An adenoma of the left main stem bronchus and carina was corrected by local resection and grafting, and it was hoped that this method may extend the limits of operability of carcinoma, particularly when it arises in the upper lobe area and involves either the carina or trachea. Bronchoscopic maneuvers following the operative procedures were carried out without fear, were done frequently through the tracheostomy, and were useful in the removal of exudate, debris, and granulation tissue.

Benign Tumors of Trachea

Waldmann [46] reported a case of multiple osteomas of the trachea which presented themselves as 1 to 5 mm. pyramidal, oval, or circumscribed densities of a cartilaginous-bony nature. In this case there was no actual connection with the tracheal rings and the lesions involved only the anterior tracheal wall, with the posterior tracheal wall being free from the smooth elevated nonulcerated excrescences. They began in the subglottic area and extended to within 2 cm. of the bifurcation of the trachea.

Dorenbusch [47] reported a case of leiomyoma of the trachea removed through an external incision in the trachea.

Foxwell [48] reported a case of solitary tracheal chondroma extending from the first to the fourth ring and arising by a broad stalk from the side of the second ring.

Tracheobronchial Trauma

Three cases of closed injury to the trachea or bronchus were reported by Richards and Cohn.[49] When there was a tentative diagnosis of traumatic rupture and uncontrollable emphysema or tension pneumothorax existed, bronchoscopy was warranted. Progressive dyspnea within 10 days after a closed chest injury demanded bronchoscopy. In the cases examined atelectasis with bronchial tear, stenosis, and granulations were observed, and in one case the dyspnea was so marked from fibrous stenosis that a long No. 3 polyethylene tube could be passed only with extreme difficulty.

Fowler [50] reported a fatal case of traumatic rupture of the left main bronchus from a closed injury to the chest. The left main bronchus was completely severed, and the proximal portion communicated directly with the pleural space, accounting for the several ounces of fluid aspirated at bronchoscopy.

A case of rupture of the right main stem bronchus from a closed chest injury which was demonstrated bronchoscopically and surgically repaired 52 days after injury was reported by Sheehy and Hopeman.[51] Complete recovery without stenosis of the bronchus followed surgical treatment.

A case of tracheoesophageal fistula due to a blast injury and repaired surgically was reported by Volk et al.[52]

Respiratory Obstruction

The features of respiratory disturbances in the newborn and young infant were summarized by Michelson.[53] Atelectasis was found to be the most frequent cause of neonatal death. Three types of tracheal obstruction in the infant were discussed: intrinsic lesions, generally caused by tumors and more rarely congenital malformations; extrinsic lesions producing compression commonly due to congenital cardiovascular defects and rarely a mediastinal cyst or tumor, and collapse of the tracheal wall associated with absent or defective cartilaginous tracheal rings. Early bronchoscopic aspiration in tracheoesophageal fistula was essential in saving the life of a case reported. Pulmonary hyalin membrane with resorption atelectasis was treated by sternal traction in 11 cases. The anomaly of

agenesis or failure of pulmonary development was rare, and the symptomatology was varied but most frequently involved the right lung.

Foreign Bodies in the Airway

Maloney [54] reiterated the clinical features of foreign bodies in the bronchopulmonary tract, calling attention to foreign bodies as a possible cause of obscure pulmonary disease that does not respond to treatment as expected. Bronchoscopy should be performed in every case with a positive history, even though the physical and roentgen findings are negative.

Withers and his co-workers [55] reported the lodgement of a foreign body in the subglottic laryngeal area as a result of direct injury. In the impact of an automobile accident a portion of the patient's upper denture was driven downward and became anchored so firmly in the subglottic larynx that it could not be removed perorally. Since the material was made of acrylic, no biting or crushing maneuver was applicable and it was necessary to extract the foreign body by means of an external thyrotomy.

Fraser [56] described a foreign body in the trachea consisting of a plastic bobbin-shaped toy whose ball-valve action produced imminent danger of asphyxiation. After tracheostomy the foreign body descended into the right main bronchus, where it became firmly impacted, and because of its smooth, round, and unbreakable surface could not be dislodged with conventional forceps introduced through the bronchoscope passed via the tracheostomy. A hook of thin wire was improvised and passed beyond the foreign body, dislodging it and bringing it up with the aid of the end of the bronchoscope to the tracheostomy opening, where removal was accomplished.

Feinmesser [57] pointed out that antibiotic treatment in cases of vegetal foreign body in the respiratory tract may reduce the infection and cause an error in diagnosis because of the diminution or disappearance of the symptoms and signs. Improvement does not lessen the indication for endoscopic removal of the foreign body, nor does it remove the necessity for bronchoscopic investigation. Occasionally endoscopy may be delayed to secure the antibiotic effect, and the intervention may be made easier after these drugs are administered.

Harris [58] described the use of the fluoroscopic intensifier for removal of a straight pin from the right lower lobe bronchus. The apparatus made possible clearness of the image with relationship to surrounding structures and good depth perception and apparently can be used much as the biplane fluoroscope or the stereofluoroscope. Removal of the foreign body was accomplished in this case by detailed guidance through this instrument.

Foreign-Body Granuloma of Lungs

Five cases of liquid petrolatum (mineral oil) granulomata of the lung in which the bronchoscopic findings were negative were presented by Wagner and his co-workers.[59] It was suggested that the pathogenesis of liquid petrolatum granulomata of the lung is due to the sclerosing effect of an endogenous fatty acid or lipoid. In four of the five cases the granuloma was situated in the upper lobes, which is in keeping with the inhalation mechanism. Two of the cases presented a mass indistinguishable from bronchogenic carcinoma; one was associated with carcinoma; one presented an atelectasis and obstructive emphysema of an upper lobe; the fifth was associated with a previously drained empyema. In all but one case there was a history of the use of nasal medication, the majority of which contained an oily base administered either as drops or in a spray.

Acute Laryngotracheobronchitis

In discussing acute laryngotracheobronchitis McEntee [60] considered the treatment in the infant so specialized and grave as to warrant special centers for its treatment. This was advocated not only to secure cooperation between the different specialities

involved but also so that nurses could become more skilled in the care of these children needing special attention.

The rationale of water vapor therapy in acute laryngotracheobronchitis was discussed by Friedman.[61] The loss of moisture from the air passages was found to be dependent upon the absolute humidity of the air breathed and not upon its relative humidity. To reduce the loss of water from the air passages not only must the relative humidity be raised to as close to 100% as possible but the temperature must also be raised to as close to 32 C as is possible for the patient's comfort. The ordinary steam kettle was considered more suitable than the mechanical humidifier for this purpose unless in addition to the humidifier other means were employed to keep the temperature elevated.

Bronchopulmonary Tuberculosis

Temple[62] found that atelectasis in pulmonary tuberculosis may be comparatively harmless or may be dangerous. Collapse of a whole lobe or of a lung may be due to bronchia occlusion, either by tuberculous stricture, obstruction by tuberculous glands from without, or accumulated secretions within the bronchus. The resulting condition was essentially one of bronchiectasis with or without the retention of caseous material in the dilated bronchi. If the tuberculous lesion in the lung was comparatively small and collapse therapy led to the appearance of atelectasis, then the pulmonary tuberculosis should be regarded as highly active. The alveoli surrounding the atelectasis became compressed by the rigid mass of active tuberculous infiltration and edema, and this type of compression atelectasis warned that the disease was advancing and sooner or later might involve the pleura with consequent empyema.

Zurik[63] analyzed 200 bronchoscopies performed on tuberculosis patients and concluded that the bronchoscopist was not fulfilling his potenital usefulness in this field so that the patients with tuberculosis could receive the full benefits of modern diagnosis and treatment. The interpretation of bronchoscopic observations was variable but was more valuable if the report included the color changes (easy bleeding), gross mucosal changes (edema, granulations, and active or healed ulceration), and distortion or stenosis. In this series 56 cases were reported as normal on bronchoscopy, yet histologic evidence of tracheobronchial disease was found in 94.6% of the resected specimens. In 39 cases reported as abnormal histologic evidence of tracheobronchial disease was found in 95%. Bronchoscopy was indicated in the presence of symptoms, physical signs, or roentgenologic findings suggestive of the disease and as a precautionary measure before any collapse therapy or resection. Serial bronchoscopy not only uncovered tracheobronchial disease after an initial negative examination but was also useful in following the progress of the disease.

A series of 79 bronchoscopies on 37 children with pulmonary tuberculosis was reported by Rubin and Harris.[64] Of the 32.4% with visible endobronchial disease, 7 had granulomata, in 4 there were deformities due to enlarged mediastinal lymph nodes, and 1 had an inflammatory stenosis about the bronchial orifice. Both the roentgen and clinical signs were unreliable in determining prior to bronchoscopy whether endobronchial disease was present. Drug therapy accomplished resolution of enlarged lymph nodes and granular mucous membrane, but formed granulomata had to be removed bronchoscopically and the exudate aspirated to restore the bronchi to a normal appearance.

Goorwitch[65] reported the development of a nontuberculous giant lung abscess in a patient with bronchostenosis secondary to healed extensive tuberculous involvement of the trachea and left main bronchus. The left upper lobe bronchus was completely obstructed by scar tissue, and the abscess was located distal to the occlusion.

A case of acquired nonmalignant bronchoesophageal fistula associated with tuberculous hilar lymph glands in a boy of 12 was

presented by Danino and his co-workers.[66] The lesion was demonstrated on bronchographic study with the use of iodized oil (Lipiodol). The only abnormality detected on bronchoscopy was reddening of the mucosa of the right lower lobe bronchus with pus exuding from all the segmental orifices. On esophagoscopy the orifice of the fistula was identified at a point 21 cm. from the upper teeth.

A case of tuberculous spondylitis of the thoracic spine with a cold abscess rupturing into a bronchus was described by Fastner.[67] Bronchoscopy revealed only copious amounts of purulent secretion in the left bronchus stemming from the left upper lobe bronchus.

Three hundred bronchograms with the use of iodized oil by vaporization were employed in tuberculous women by Simonin and his associates.[68] Only 27 of the bronchograms were considered normal, and the commonest abnormality seen in 210 cases was moniliform dilatation. From operative specimens three types of bronchial lesions were found: tuberculous inflammatory bronchitis, discrete caseous bronchitis, and intense caseous bronchitis. It was concluded that pulmonary tuberculosis was accompanied early and in the majority of patients by lesions of the bronchi of the diseased segment due to involvement of the elastic fibers, making the changes irreversible. It was recommended that all tuberculous patients be examined by the harmless thin-layer iodized oil method of bronchography described.

Perry[69] pointed out that bronchography was a dangerous investigation in patients with pulmonary tuberculosis when a medium containing iodine was employed because the iodides tend to activate tuberculosis. Both iodized oil and the newer medium Dionosil are iodide preparations. Two patients were presented in whom tubercle bacilli appeared in the sputum after bronchography with oily media. In three others tuberculous bronchopneumonia was precipitated by the investigation.

Forgacs[70] performed a series of 120 bronchograms with Dionosil in patients with pulmonary tuberculosis. The procedure was found to be safe and was employed as a preliminary investigation to major surgical procedures, especially resection. It was of considerable help in localizing the precise site of the disease and in demonstrating unsuspected bronchial abnormalities not seen on plain roentgenograms. There were no bronchographic features peculiar to active tuberculosis noted, and no indication of the state of healing of the disease was provided by bronchography.

A series of 240 bronchograms with the use of Dionosil Oily in 174 patients with pulmonary tuberculosis was recorded by Holden.[71] Bronchography was carried out as rapidly as possible, and the medium was allowed to remain in the lungs for a minimum length of time. The medium was easy to use, relatively nonirritating and nontoxic, and well tolerated. Adequate contrast and quick elimination were obtained, and complications were unusual.

Shafran and Kavee[72] believe there has been an increase in the frequency of coexisting pulmonary tuberculosis and bronchogenic carcinoma, partially accounted for by the shifting incidence of tuberculosis into the older age groups. In a period of one year, six cases were encountered and the primary factor in the diagnosis was the roentgenologic appearance. When confirmatory bronchoscopic, histologic, and cytologic evidence was lacking, exploratory thoracotomy was performed. Any patient with roentgenologic regression of tuberculous densities on rest and adequate chemotherapy accompanied by simultaneous growth or failure of other noncalcific lesions to improve was suspected of having both carcinoma and tuberculosis of the lung.

The difficulties in the diagnosis of coexistent bronchogenic carcinoma and active pulmonary tuberculosis were discussed by Wayl,[73] who reported three cases, one of which was in a 27-year-old man. Atelectasis, or pain in the absence of pleural involvement, accompanied by wheezing and

hemoptysis was more frequent in cancer than tuberculosis. Early bronchoscopy with repeated cytological examination of the secretions should be employed to detect cancer in cases of pulmonary tuberculosis.

Bronchography

Flake and Ferguson[74] described their method of tracheography and bronchography in infants and children. The diagnostic value was unquestioned and can be done at any age, preferably under anesthesia. Successful technique required the services of an endoscopist in cooperation with an anesthetist and roentgenologist to handle the patients speedily and skillfully. The chief symptoms requiring these studies were cough, stridor, wheezing, or roentgenologic signs of obstruction or other changes of a chronic nature.

A simple technique for bronchography was described by Thomas.[75] After anesthetizing the larynx and trachea with 2% cocaine solution, iodized oil was instilled into the tracheobronchial tree by a curved laryngeal cannula and the patient positioned on the x-ray table to secure films.

A rapid method of performing bilateral bronchography was described by Gianturco and Miller.[76] Any contrast medium can be used, but Dionosil Oily was preferred. The contrast medium was injected through an intratracheal catheter as rapidly as possible (four to six seconds) in the lateral decubitus, with an upward tilt of the table of about 10 degrees. The catheter remained above the carina, and the rapid injection filled the dependent main bronchi and covered the openings of all its branches at once. As soon as the injection was finished the patient was positioned and roentgen films taken.

Fifty-nine patients with tuberculosis were examined bronchographically with water-soluble contrast media by Rengarts,[77] who found Ioduran B * easier to work with and productive of better contrast than Xumbradil Viscous B. A wet bronchial tree was found to be advantageous, and rapid emptying with no residual or side-effects was characteristic. The success of water-soluble media depended upon adequate anesthesia. They mixed with body fluids and gave uniform bronchial outline.

Oily Dionosil was employed in 30 bronchograms by Wisoff and Felson.[78] No significant difference between Dionosil and iodized oil in contrast density or detail was seen on the roentgenograms. The one advantage noted was rapid disappearance of the Dionosil on the chest roentgenogram, and in 17 cases submitted to lung resection no gross or microscopic damage to the lung could be demonstrated.

Zizmor[79] reported two cases of bronchogenic carcinoma with normal chest roentgenograms in which the diagnosis was first suspected by bronchographic examinations.

The bronchographic findings in the lung periphery in chronic bronchitis were correlated with the pathologic changes by Reid.[80] Incomplete peripheral filling indicated stenosis and often obliteration of the bronchiolar lumen and did not necessarily mean that the technique was inadequate or that the bronchioles were plugged with secretion. The second abnormality noted was the presence of peripheral pools when radiopaque material was held in a dilated bronchiole, usually immediately proximal to a point of obliteration. The third abnormality was a spiked outline which somewhat resembled the normal bronchiolar pattern but was coarser. These spikes resulted from the filling of terminal bronchioles which were dilated proximally to points of obliteration.

Bronchiectasis

Mulder and Hers[81] advanced the theory that fetid mucopurulent bronchitis with multiple (anaerobic) bacterial flora in fetid sputum is correlated with the presence of

* A highly viscous preparation (50% diethanolamine salt of diiodopyridone-N-acetic acid and 2.5% sodium salt of cellulose glycolic acid ether). Xumbradil Viscous B has basically the same chemical formula as Ioduran B but is only approximately half as viscous as Ioduran B.

metaplastic squamous bronchial epithelium. The theory was borne out by the findings of metaplastic epithelium in 9 out of 10 cases with fetid sputum upon bronchoscopic biopsy and in the extirpated lobes of 3 other cases. The squamous epithelium did not cover the entire surface of the bronchi in these cases but appeared focally and not especially at the carina. Of practical import was the possibility that the inexperienced observer may mistake the squamous epithelium in the sputum for tumor cells.

An evaluation of the surgical treatment of bronchiectasis in 30 children was summarized by Cooley and his associates,[82] who found that the results of surgical treatment were not as good in children as those in a series that included patients of all age groups. An increase in the instance of postoperative complications, particularly retention of secretion, was noted in children, and they also showed an increased tendency for the postoperative development of new bronchiectasis. Incomplete development or increased susceptibility to infection may be a contributing factor in the different results obtained in the treatment of this disease in children and adults.

Mycotic Infections

Ferguson[83] pointed out that mycotic pulmonary diseases often are confused with other pulmonary lesions, particularly tuberculosis and cancer. Early diagnosis is now important because specific treatment has been devised for some forms of mycotic disease. In all pulmonary cases where a clear-cut diagnosis can not be reached the possibility of a fungus infection must always be considered. Pulmonary mycotic diseases usually closely resemble other pulmonary ailments and there are no specific features which identify them, so that repeated and continued laboratory studies are necessary in this respect. Of a total of 117 patients with infections caused by actinomycosis, 15 had pulmonary infection, 10 with involvement of other structures as well and 5 of the lungs alone. Seven were diagnosed by culture or biopsy of the draining sinuses and five from culture of sputum. Both aerobic and anaerobic forms of actinomycosis responded to antibacterial therapy. Nine instances of Nocardia infection were present, three of which were pulmonary in origin. Only three instances of pulmonary Aspergillus infection were noted, and positive sputum cultures established the diagnosis in each instance. One hundred three cases of blastomycosis were observed, and twenty-nine of these were pulmonic. The diagnosis was made in 22 cases as follows: 8 by sputum culture, 7 by pus from a draining sinus, 2 by laryngeal biopsy, 2 by lung resection, 2 at postmortem, and 1 by an associated skin lesion. Blastomycosis was successfully treated by 2-hydroxystilbamidine therapy. Six instances of coccidioidomycosis were found, of which two were pulmonic. Twenty-two had nodular pulmonary densities and seven had cavities, but when the condition was progressive it responded poorly to any known therapy. Eight cases of cryptococcosis were seen, two of which were pulmonic in origin and both were complicated by brain and meningeal infection. The diagnosis was made by spinal fluid examination. Eight cases of probable pulmonary histoplasmosis were reported, and although the sputum culture was negative for the organism, all skin tests were strongly positive to histoplasmin. There were 62 instances of pulmonary infection due to monilia, most of which were secondary invaders.

The close resemblance of mycotic infections of the lungs to pulmonary tuberculosis was emphasized by Penta.[84] The commoner forms of pneumonomycosis—moniliasis, torulosis, blastomycosis, actinomycosis, coccidiosis, histoplasmosis, sporotrichosis, and aspergillosis—were described in detail. Mycotic infections occur oftener than is generally suspected, and in obscure pulmonary disease this possibility should always be considered. The diagnosis is made by isolation of the offending fungus. Antifungal therapy may be beneficial, and in

suitable cases pulmonary resection may effect a complete cure.

A series of 67 cases of pulmonary histoplasmosis was reported by Puckett.[85] Many of these cases were diagnosed clinically as tuberculoma or coin lesion of the lung. In approximately half of the series the patients were asymptomatic and the lesions were only found on routine chest roentgenograms. The lesion mimicked both tuberculosis and neoplasm, and special care in pathogenic examination was necessary in granulomatous lesions to establish the diagnosis.

Negative bronchoscopic findings were reported by Falkmer and Wising[86] in a fatal case of bronchopulmonary moniliasis.

A case of North American blastomycosis in the presence of bronchogenic carcinoma was reported by Abraham and Luddecke.[87]

Lung Abscess

Waterman and his co-workers[88] were of the opinion that bronchoscopy should be performed in every case of lung abscess or suppurative pneumonia within the first 24 hours, and in a high percentage of their 268 cases it was not necessary to resort to surgical measures other than bronchoscopy. From the diagnostic standpoint malignancy must be ruled out as well as other pathologic disorders, and valuable information regarding the inflammatory reaction in the bronchus was gained. Another function of bronchoscopy was to obtain specimens uncontaminated by mouth secretions for organism and antibiotic sensitivity determinations. From the therapeutic standpoint an efficient bronchoscopy helped evacuate accumulated and obstruction secretions, permitted shrinking of the mucous membranes by topical applications, and increased cough, thereby making elimination of secretions more effective. The favorable effect of bronchoscopy was manifested within a few days and lasted for 7 to 10 days. If no improvement followed the second bronchoscopy the therapy was changed. Frequent bronchoscopies were considered unnecessary and frequently harmful.

Kessler and Skinner[89] found bronchoscopy useful in the study of all cases of lung abscess to localize the bronchus draining the abscess and detect the presence of a foreign body or other obstruction, such as a tumor. Secretions taken directly from the involved area at bronchoscopy were examined for tubercle bacilli and tumor cells, and antibiotic sensitivity tests were performed on the causative pyogenic bacteria. Medical treatment consisting of bed rest, transfusions, postural and bronchoscopic drainage together with large doses of antibiotics by parenteral and aerosol routes was recommended first and continued as long as improvement was definite and prompt.

Gittens and Mihaly[90] advocated bronchoscopy in acute pulmonary abscess when the etiology was obscure, the response to conservative therapy slow, or when foreign body or malignant growth was suspected. Bronchoscopy was found to be valuable both in diagnosis and therapy.

Pulmonary Scleroderma

Dawson[91] reported a case of pulmonary scleroderma manifested on the roentgenogram by diffuse reticulation with many small cysts at the lung base.

Amyloid Disease

Gordon[92] described a case of primary amyloid deposits in the bronchus which bronchoscopically resembled an inoperable carcinoma. At bronchoscopy on several occasions there was noted distortion and widening of the carina with narrowing of the left main bronchus. A swelling extended along the medial wall of the left main bronchus, and irregular tissue removed from the left upper lobe bronchus revealed the presence of amyloid deposits. Autopsy demonstrated that the amyloid was confined to the submucosa, muscularis, and adventitia of the left and right main bronchi.

Aerosol Therapy

Denton[93] outlined the physiologic basis, rationale, and clinical experience derived

from continuous nebulization therapy in bronchopulmonary disease. The basic mechanical principle was the same as that of the standard aerosol therapy whereby microscopic droplets of fluid were added to the inspired atmosphere. It was valuable in controlling the factors often responsible for obstruction and deficient pulmonary ventilation, i. e., retained secretions and edema. Besides the moisture additional therapeutic effect was obtained by the addition of specific medicaments which act locally on the mucous membranes. The solutions used were propylene glycol (to lessen evaporation), bronchovasoconstrictors, wetting agents, and, rarely, antibiotics. The clinical application of this method was found advantageous in acute laryngotracheobronchitis, foreign body, bronchiolitis, and aspiration pneumonitis in children, after tracheotomy and in medical and surgical pulmonary diseases in adults.

Marchetti [94] described a method of continuous administration of drugs into the tracheobronchial tree for prolonged bronchoclysis in the treatment of bronchial suppurations. After local anesthesia of the larynx and tracheobronchial tree a rubber catheter was introduced into the trachea and a small polyethylene tube was positioned through the catheter. After it was in position the catheter was withdrawn and the polyethylene tube was brought out through the nose. Drop-by-drop infusion was used at the rate of five drops per minute and was tolerated by patients as long as 30 to 60 days. During this time the patient slept and ate with little inconvenience. The method usually used to instill the drug was an hour of instillation (until the bronchial mucosa became saturated with the drug) and a half hour of rest. Except for red rhinopharyngitis the procedure was apparently tolerated without production of any specific lesions.

Endoscopic Treatment of Bronchial Fistula

Guida [95] treated 14 cases of postoperative fistula formation after pulmonary resection for tuberculosis by endoscopic cauterization with sodium hydroxide. Twelve cases closed, and in only two patients was there clinical evidence of fistula, both of whom were in poor general condition.

Benign Tumors of Bronchus

Hochberg and Schacter [96] considered bronchoscopy a valuable adjunct in the diagnosis of endobronchial tumors, but the differentiation between a benign and malignant tumor by this method was unreliable. In approximately half of the cases of benign tumors of the lower respiratory tract visualization of the growth was accomplished endoscopically and the diagnosis was made on histopathologic study of representative tissue. Adenoma of the bronchus constituted approximately 60% of all benign tumors occurring in this area. In the treatment of benign tumors bronchoscopic removal was inadequate in most cases but was valuable in palliative therapy and to permit evacuation of the contents of the lung distal to the obstructing tumor.

Bronchoscopy has been condemned in cases of pulmonary air cysts because of the theoretical possibility of rupture due to increased intrabronchial pressure and strain, but Dugan and Hardy [97] have not seen this development during or following this procedure. Bronchoscopy was employed in all cases in which secondary bronchial obstruction was suspected to rule out foreign-body or other bronchial lesions contributing to the development of air cyst.

Two cases with various manifestations of papillomatosis of the lower respiratory tract successfully treated by endoscopic removal were presented by Schaff and Thomson.[98] One patient had papilloma of the right main and upper lobe bronchus with multiple recurrences for 17 years. The other patient developed multiple lesions in the trachea and major bronchi on the left five years after the occurrence of laryngeal papillomata. The etiology remains obscure, but there is more evidence in favor of the viral origin than local implantation of desquamated papilloma cells.

McCall and Harrison[99] reported a case of intrabronchial lipoma. On bronchoscopy a smooth globular tumor was observed almost completely blocking the left main bronchus just distal to the carina. Suppuration and bronchiectasis were present distal to the obstruction.

A case of intrabronchial leiomyoma which was indistinguishable from adenoma and fibroma of the larger bronchi in its gross appearance was presented by Hirose and Hennigar.[100]

A case of granular-cell myoblastoma of the bronchus was reported by Ramsey.[101] The clinical findings suggested bronchial adenoma, and on bronchoscopic examination a mass, approximately 2 cm. in diameter, was observed in the left main stem bronchus arising from the anterior medial wall. Because of the presence of bronchiectasis of the right lung, it was decided to remove the tumor in the left bronchus bronchoscopically. Profuse bleeding prevented total excision; but the majority of the tumor was removed, so that there was no appreciable obstruction of the bronchus after the completion of the four bronchoscopic treatments necessary to obtain this.

Sarcoma of Bronchus

A case of primary fibrosarcoma of the bronchus with negative bronchoscopic findings was reported by McEachern et al.[102]

Bronchogenic Carcinoma

The greatest value of bronchoscopy in bronchogenic carcinoma was in determining technical resectability rather than obtaining biopsy material, according to Overholt and Bougas.[103] It proved to be the most precise gauge of the tumor's extent within the major air passages. Contralateral bronchial or tracheal involvement was a finding which contraindicated resection. Fixation of the mediastinum suggested invasion of mediastinal lymphatic system, while fixation of other portions of the tracheobronchial tree generally meant carcinomatous invasion of paratracheal lymph nodes, but in some cases immobility was produced by inflammation. Enlargement of mediastinal nodes as evidenced by the condition of the carina was not considered an absolute contraindication to successful resection but only a presumptive contraindication. A drop in the bronchoscopic verification by tissue removal from 66% prior to 1941 to 22% after 1949 was noted. During the same period verification by exploratory thoracotomy rose from 18% to 57%, indicating the value of this procedure in any questionable lesion and the importance of early surgical exploration. In a group of 462 cases of verified primary pulmonary carcinoma, 348 bronchoscopies were performed and 104 pathologic diagnoses made. Although 76% of all patients were bronchoscoped, only 30% of the bronchoscopies gave a positive result for malignancy. Seventy-four per cent of the lesions in this group were centrally located near the hilum, and few were discovered by bronchoscopy in the proximal bronchi or in the peripheral region. The authors stated that it was not always wise to remove a specimen at bronchoscopy when the lesion was visible because of the danger of serious intrabronchial hemorrhage, and recent practice has been to omit biopsy on those patients who are to undergo immediate thoracotomy and have an uninvolved trachea, carina, and contralateral bronchus. The least possible time should elapse between bronchoscopy and thoracotomy, and in some cases it was suggested that a preoperative bronchoscopy be performed under topical anesthesia, followed immediately by thoracotomy, should no unexpected contraindications be discovered. Doubtful resectable cases were scheduled for separate bronchoscopic evaluation before surgery was decided upon.

In a series of 120 resected cases of bronchogenic carcinoma bronchoscopy was performed in 88 by Alvarez[104] and was positive by direct or indirect signs in 42.3%. When bronchoscopy was positive the diagnosis was confirmed, but negative bronchoscopy did not rule out carcinoma as established by clinical and radiological means. From the standpoint of resection negative

bronchoscopy was favorable, for it usually meant that the cancer was peripherally located. Adenocarcinoma located almost exclusively peripherally yielded 13.8% positive bronchoscopic findings as against the epidermoid type located near the hilum with 62.2% positive bronchoscopies.

Toft [105] achieved a positive preoperative diagnosis of carcinoma by biopsy or cytologic examination of secretion at bronchoscopy in approximately 60% of a total of 355 cases of bronchogenic carcinoma, and a strong suspicion was present in another 22%. Positive diagnosis was obtained at bronchoscopy in 61.4% of the squamous-cell type, 61.2% of the anaplastic type, and 33.3% of the adenomatous type. In the latter the low percentage was attributed to the frequent peripheral location. Suspicious microscopic diagnosis was made in an additional 21.2% of the squamous-cell type, 22.5% of the anaplastic type, and 38.1% of the adenocarcinomas. Of the entire group established or suspicious carcinoma was present in 45% on biopsy and 52.5% by cytologic examination.

McDonald [106] reported positive pulmonary cytology in 1600 cases. In 76% of these the positive diagnosis of malignant cells was made from examination of sputum, while in 39% it was made from bronchial secretions, with some cases having both sputum and bronchial secretions positive. A false positive diagnosis was obtained in 1.7% of the entire group. The false negative error was between 30-35%. The results were reported either as positive or negative on a purely objective basis.

Gibbon and his associates [107] reported a consecutive series of 617 cases of carcinoma of the lung. By means of bronchoscopy a positive histologic diagnosis was established in 65% of the cases—in 23% by biopsy and in an additional 42% by cytologic examination of bronchial secretions.

Shabart [108] found 42% positive bronchial biopsies in 108 proved cases of bronchogenic carcinoma and 50-53% positive Papanicolaou smears in 70 proved cases. In 64 proved cases 44% positive Papanicolaous and 48% positive biopsies were obtained, making a total of 75% positive reports by either Papanicolaou smear or biopsy. In 24% neither the secretion nor the biopsy was positive for carcinoma.

McCormack et al.[109] reviewed 602 patients in whom cytologic examination of bronchial swabbings was carried out for the diagnosis of cancer of the lung. Two hundred patients were regarded as clinically having primary carcinoma of the lung, and the cytologic examination was positive in 47%. With the combination of bronchoscopic biopsy and cytologic study two-thirds of the cases were diagnosed positively, and no false positives were encountered. In the method described a technician specially trained in cytologic technique was present at the time of the bronchoscopy. Material was obtained through the bronchoscope, either by aspiration, direct swabbing, or biopsy. When washings were made the aspirated fluid was transferred to containers for centrifugation and study of the sediment. Small sponge pledgets attached to applicators were used to swab the suspicion-arousing areas within the trachea or bronchus after they had first been moistened with saline solution. While in the wet state the specimens were prepared using two wet smears and two smears of the rapidly fixing type. Although there may be advantages to the wet suspension, the cytologic appearances differ so much from that of the fixed specimen that an intimate knowledge of this difference is needed by the cytologist for accurate interpretation.

Spjut and his associates [110] reviewed 501 confirmed cases of pulmonary cancer and found bronchial washings consistently more productive of positive cytologic results than sputum examinations. Bronchial secretions were positive in 31.6%, and cancer in the lower lobes yielded a higher percentage of positive results than in other localities. Cytologic diagnosis was also valuable in the diagnosis of peripheral lesions. Lesions 2 to 3 cm. in diameter and those larger than

7 cm. produced the highest percentage of positive smears, while cases with positive hilar and bronchopulmonary nodes yielded a higher percentage of positive bronchial washings than those with negative nodes. Positive smears were obtained in 56.1% of the operable cases and 59% of the inoperable cases. The type of cancer was accurately diagnosed from the smears in 79.3%, and cytology was the only positive tissue finding in 59 of the operable cases.

Gregg and his associates [111] increased the number of neoplastic cell clumps in specimens obtained from peripherally located pulmonary carcinoma and increased the positive results by means of "placer mining." The technique consisted of abrasion of the bronchial mucosa in the suspected area with a suction tip to separate small flecks of mucosa, irrigation by means of a jet of saline solution, and aspiration of the material into a collection vial. Of the 30 patients with malignant disease, there was evidence of neoplasm grossly by bronchoscopic examination in 70% with bronchial washings strongly suggestive or diagnostic in 63%. There were five cases in which there was no evidence of lesion and the aspirated specimen was not diagnostic. Of the cases examined the presence of a lung cancer was suggested by bronchoscopic findings and examination of bronchial washings in 80%.

McBurney and his associates [112] reported 29 cases of asymptomatic bronchogenic carcinoma discovered by roentgenologic study. Bronchoscopy proved negative in 20 of 22 cases, and cytologic examination gave negative results in 20 of 23 cases. In three cases the diagnosis was made by the finding of malignant cells in the sputum. In every case the lesion was resectable.

Multiple Malignant Lesions

In 2502 cases of lung cancer Cahan [113] found 81 instances in which the lung tumor was one of two or more separate primary malignant growths, making an incidence of 3.2%. In these 81 cases there were 34 instances in which the cytology of the bronchial secretions was positive. In six exfoliative cytology alone established the diagnosis of cancer. In two others the cytology as well as biopsy was positive, and in four cases the cytology was negative. Bronchoscopic biopsy helped to clarify the differential diagnosis in two cases by distinguishing squamous carcinoma from Hodgkin's disease and oat-cell carcinoma from squamous carcinoma of the extrinsic larynx. When the cells obtained by exfoliative pulmonary cytology revealed the same histologic structure as the known primary cancer elsewhere, it was impossible to know whether metastasis was present. The finding of carcinoma by the cytologic method helped to establish the true nature of equivocal pulmonary shadows, as demonstrated in 5 of 18 cases in which solitary metastatic foci rather than primary carcinoma of the lung were found. In one case the correct diagnosis was made when the first carcinoma was adenocarcinoma and the cytologic picture of the bronchial washings was squamous carcinoma.

Two concomitant but independent growths in the tracheobronchial tree were reported by Bolstad.[114] On bronchoscopy a fungating lesion was found arising from the anterior surface of the trachea about 1 cm. proximal to the carina. A separate mass was observed protruding from the orifice of the right middle lobe bronchus and producing obstruction in this area. Although metastasis of bronchogenic carcinoma to the opposite lung is not rare, this case presented two primary tumors in the tracheobronchial tree.

A case of concurrent primary leiomyosarcoma and carcinoma of the bronchus was reported by Rosenberg et al.[115] Bronchoscopy revealed a mass 2 cm. below the carina partially obstructing the right main bronchus, but tissue removed at three different times was diagnosed only as necrotic tissue. The carcinoma was situated close to the sarcoma, but no histologic continuity could be found.

Esophagology

In reviewing progress in esophagology Baker and Flood[116] stated that improvement has been the result of the continued efforts of all branches of medical sciences, including radiology, anesthesiology, anatomy, and physiology. Although the problems encountered in esophageal disease remain varied, conservative therapy was always carried out initially. If a functional esophagus was unattainable by this method surgery was employed. Postsurgical problems often developed and had to be treated later by the esophagologist and gastroenterologist.

The role of the esophagologist in aiding the surgeon in the diagnosis and management of esophageal lesions and a method of retrograde dilatation of the esophagus through the esophagoscope were discussed by Hoover.[117] A case in which a dilated hypopharynx, manifested by dysphagia, occurred secondary to obstruction from the cricopharyngeus was relieved by dissection of the cricopharyngeus muscle. Complete relaxation of the cricopharyngeus and pharyngeal constrictor paralysis secondary to unilateral 10th cranial nerve paralysis was observed in one case. Operation upon small pharyngoesophageal diverticula was recommended using a procedure designed to decrease the dilation of the hypopharynx from bag-like to funnel- or V-shape.

Hoover[118] recorded his personal clinical observations on nonneoplastic conditions of the pharynx and cricopharyngeus area, described a method of retrotransesophageal dilatation, and emphasized several points in esophagoscopy. The following points were covered: the history and physical condition of the patient; roentgen examination of the pharynx and esophagus together with the cervical spine for exostoses before esophagoscopy; preparation of the esophagus by lavage; intubation; inflation of the esophagus during examination; the possibility of multiple lesions in the esophagus; the use of antibiotics to prevent infection following trauma. His method of retrograde dilatation of the esophagus through the esophagoscope was thoroughly described and an illustrative case reported. Attention was called to the resulting relaxation of the cricopharyngeus muscle in vagus nerve paralysis. Dysphagia due to cricopharyngeal spasm or hypertonicity was described as a clinical entity, and relief was obtained by local interference with the vagus nerve supply to this muscle by careful surgical dissection of this region. In the surgical treatment of pharyngeal diverticuli the importance of repairing the muscular pharyngeal wall and infolding the hypopharynx to correct the dilatation was emphasized. In two cases true stricture formation due to fibrosis at the cricopharyngeal area was the etiologic factor in the production of a pulsion pharyngeal diverticulum. The technique of the operative procedure for both small and large diverticula was described, and tailoring of the dilated hypopharynx to a funnel to meet the anatomical variations of large diverticula was advocated. Operative treatment was performed for small diverticula, and although the surgically opened pharynx may heal without the rest and protection of tube or intravenous feedings this procedure was not recommended.

Dysphagia

Sclerosis of the aorta was described as the cause for dysphagia in seven cases by Keates and Magidson.[119] All the patients were elderly women with arteriosclerotic and hypertensive heart disease in whom other causes for dysphagia were ruled out by esophagoscopy.

A case of tetanus with the presenting symptom of dysphagia was recorded by Stephenson and Blacklay.[120]

Wolff et al.[121] reported three cases of thrush esophagitis accompanied by swallowing incoordination and aspiration pneumonia effectively treated with hydroxystilbamidine.

Atresia of Esophagus

Paradoxical hematemesis occurred in the case of esophageal atresia reported by

Lecutier.[122] Vomiting can occur in esophageal atresia with a fistula from the lower segment into the trachea and thence into the mouth, provided there is material in the stomach to vomit.

Following surgical correction of esophageal atresia Clatworthy[123] found that four of the seven surviving patients subsequently required dilatations. Dilatation was accomplished perorally in the majority, and rarely was retrograde dilatation through a gastrostomy opening used.

Esophageal Obstruction

In Puestow's[124] opinion, conservative management of occlusive disease of the esophagus resolved itself into correcting stenosis due to three major underlying processes: (1) functional occlusion without organic change, (2) functional occlusion with organic change, and (3) chronic stenosing esophagitis. When the condition was considered functional, as in cardiospasm, an expansile type of balloon dilator, such as the Plummer bag, was advocated, but if the stenosis was due to constriction from scar tissue a rigid type of graduated dilator was needed, such as the metal olive-tipped bougies. In cardiospasm complete symptomatic relief usually followed a single dilatation even though no demonstrable organic change was noted on the roentgenogram. In chronic fibrotic esophageal stenoses the passage of a dilator must be over a guide, such as a swallowed string or fine wire. In these cases as long as the smallest opening was maintained the patient could be adequately nourished.

Three uncommon cases of subcarinal mediastinal granulomas producing esophageal obstruction were recorded by Garamella et al.[125] Compression stenosis was observed roentgenologically and esophagoscopically, and the etiologic factor was mediastinal extension from caseous bronchial glands presumed to be tuberculous.

Esophageal Perforation

In the management of instrumental perforation of the esophagus Howard[126] listed five important points: (1) prophylaxis through gentleness; (2) realization that the hypopharynx and esophagus of the aged are not resistant to trauma; (3) treat any post-instrumentation complaints promptly; (4) initially treat perforations by hospitalization, Trendelenburg position, and intravenous or tube feeding; (5) surgical drainage if conservative treatment is unavailing or the injury seems too large for conservative treatment.

Adkins[127] considered esophageal perforations as surgical emergencies whether spontaneous or caused by burns, foreign bodies, or instrumentation. Early diagnosis is essential to successful treatment consisting of mediastinal drainage and antibiotic therapy. Closure of the perforation and mediastinal drainage were advocated if the condition was recognized within 24 hours, unless contraindicated by preexisting disease.

Overstreet and Ochsner[128] reported 13 patients with traumatic perforation of the esophagus that were operated upon as soon as possible after the diagnosis was made and resuscitative and supportive therapy begun. Ten of the patients received primary repair with excellent results except for small temporary fistulas that developed in three cases. The duration of the perforation prior to repair in those developing fistulas was 44, 24, and 28 hours, respectively. The time interval from perforation to surgery ranged from 1½ to 44 hours. Of the eight endoscopic perforations, five occurred immediately below the cricopharyngeal sphincter on the posterior esophageal wall. One occurred through a stricture immediately above the esophageal hiatus, and in the two patients in whom drainage alone was done the site was in the cervical or upper thoracic esophagus but the opening was not visualized. All four of the penetrating esophageal injuries occurred in the cervical segment. One patient died following mediastinotomy and drainage for massive infected pleural effusion and tension pneumothorax 28 hours after perforation. Rapid and relatively uncomplicated recovery from this

previously highly fatal accident can be obtained by prompt diagnosis, rapid resuscitation, surgical exploration with debridement of contaminated fascial planes, repair of the esophageal defect, and vigorous postoperative supportive therapy.

Functional Esophageal Disturbances

Lindsay [129] divided functional disturbances of the upper swallowing mechanism into those affecting musculature, innervation, and psychic control. Objective evidence of the swallowing mechanism was obtained by fluoroscopy and roentgen films, and any weakness or asymmetry of the action of the constrictor muscles and the cricopharyngeus was confirmed by x-ray examination. It was not possible by this means to detect increased tone or spasm of these muscles. Those diseases which produced changes in the action of the musculature included myasthenia gravis, scleroderma, dermatomyositis and myasthenia associated with tuberculosis, thyrotoxicosis, and advanced senility. Both motor and sensory disturbances of innervation occurred in lesions of the 10th cranial nerve. The motor disturbances consisted of peripheral neuritis, jugular foramen syndrome, lesions affecting the nerve as it leaves the bulb and enters the jugular foramen, and intracranial lesions. Psychic factors producing disturbances of the swallowing mechanism were frequent and varied in severity, and treatment was directed to the basic psychic disturbance rather than to the local manifestation.

Foreign Bodies in the Food Passages

Ausband and his associates [130] reported two cases of a marble in the esophagus of a young child. In a 3-year-old child the marble was lodged above the cricopharyngeus, but attempts to remove it endoscopically initially were not successful, since forceps of the proper size were not available and the foreign body was pushed down below the cricopharyngeus muscle. Subsequently under fluoroscopic guidance a Johnson stone basket was used to encircle the marble and removal was accomplished without difficulty. In the second case the marble was also removed but more difficulty was encountered due to the fact that the foreign body was lodged in the lower esophagus and the esophageal folds were more redundant and flaccid. Because of the danger of breaking one of the wires with the complication of perforation of the esophagus, the wire-type Johnson stone basket was discarded in favor of the nylon basket, which works on the same principle but provides greater safety.

Richardson [131] presented a case in which linear calcification in the lamina of the cricoid cartilage simulated a foreign body in the esophagus even after removal of a chicken bone.

Using a short esophageal speculum Schaff [132] removed an embedded sandbur from the left pyriform sinus without difficulty.

Miller and Godfrey [133] reported a case in which cooked meat became impacted in the esophagus and was removed by the administration of trypsin. In achalasia associated with retention of a large amount of food mechanical removal through the esophagoscope proved tedious but was readily accomplished by the instillation of trypsin into the esophagus. The use of an air-vent suction system in conjunction with trypsin offered an additional advantage in the prevention of aspiration of digested food into the lungs.

Equen et al.[134] reported the removal of an open safety pin in the esophagus of an elderly patient by means of an esophagoscope, forceps, and magnet.

In three cases Brizzolara [135] used the Alnico magnet to extract foreign bodies from the gastrointestinal tract.

Finerman [136] presented the case of a child of one year of age with 5 open safety pins in the stomach and at least 12 needles variously distributed in the respiratory and alimentary tracts. It was pointed out that except for foreign bodies in the larynx or trachea metallic foreign bodies in the re-

spiratory or alimentary tract do not demand emergency extraction. In this case perforation without serious complication occurred when the needles migrated through the walls of the gastrointestinal tract. All the other foreign bodies passed spontaneously except one safety pin and one needle. An unsuccessful attempt was made to remove the safety pin perorally, and it was removed by surgical gastrostomy.

An unusual complication of a foreign body of the esophagus was reported by Barton [137] in which there was long delayed retroesophageal inflammatory reaction with recurrent mediastinitis 18 years after the lodgement of the foreign body. In spite of antibiotic and surgical techniques the patient never recovered from the complication, and the final outcome was the intervention of a fatal bulbar poliomyelitis.

Pharyngoesophageal Diverticulum

A series of 339 cases of pharyngoesophageal diverticula treated by one-stage diverticulectomy was reviewed by Mortensen et al.[138] The mortality rate was less than 1%, and the average hospitalization period was 9.1 days. Of the 154 cases followed, 8 had persistent symptoms—3 with recurrence of the diverticulum, 2 with postoperative constriction at the operative site, 2 with unexplained dysphagia, and 1 with unilateral vocal cord paralysis. The one-stage operation offered many advantages over the two-stage procedure and was considered the procedure of choice for most, if not all, patients with this lesion.

Fifty-three patients with pharyngoesophageal diverticula were operated upon by Jackson and Norris,[139] who described the symptoms and surgical technique. The operation was performed in one stage, and the esophagoscope was used in certain parts of the operation to aid exposure and identification of the sac. One postoperative death occurred, and the average period of hospitalization was 14 days. One case in which esophagoscopic guidance to determine the site of amputation and closure of the sac was not used developed a stenosis that required several esophagoscopic dilatations before cure. In six cases a left recurrent laryngeal paralysis developed and several recovered. No case developed a mediastinitis, although there was one delayed wound healing from superficial infection. The value of the one-stage esophagoscopically guided pharyngoesophageal diverticulectomy was reconfirmed by the results reported.

Johnston [140] reported a case of surgical removal of a diverticulum of the hypopharynx. A surprising shift of the pouch from right to left was noticed during the year prior to its removal and was explained on the basis of expansion in the direction of least resistance. Because of the uncertainty of the exact location of the pouch, preference was expressed for the transverse incision and the desirability of an esophagogram or endoscopic examination immediately prior to surgical treatment. The single-stage surgical extirpation was used, and the patient made a satisfactory recovery.

Kesseler and Maier [141] reported a case of a perforated esophageal diverticulum which was treated by immediate diverticulectomy. The perforation occurred at esophagoscopy, and operation was performed within a few hours before an acute inflammatory process interfered with the esophageal repair.

Riberi and his co-workers [142] reported a case of intraepithelial carcinoma arising in a pharyngoesophageal diverticulum discovered on histologic examination of the excised sac.

Esophagitis

The etiology, incidence, and morbid anatomy of 116 patients with severe digestion esophagitis were discussed by Peters.[143] The terms digestion, peptic, and reflux esophagitis were used interchangeably and were defined as inflammation of the esophagus directly due to digestion by gastric secretion. The disease was found to be common and many cases were listed, including those due to both extrinsic and intrinsic

factors. The pathologic finding was chronic digestion of the squamous portion of the esophagus irrespective of the occurrence of ulcers in the gastric lined portion. At any stage of the disease it was thought that death could follow perforation into the mediastinum, pleura, or pericardium and there was general liability to inhalation bronchopneumonia. The patients also tended to become anemic from intermittent or chronic oozing from the raw surfaces. Long-continued cases developed strictures which were often quite severe.

The histopathology of subacute erosive esophagitis was summarized by Palmer [144] from biopsy specimens obtained from 61 patients. Disease was localized largely to the lamina propria mucosae, and serial biopsy specimens removed at variable intervals from 12 patients revealed static histologic findings. Since the epithelial layer itself was normal or secondarily diseased from inflammatory exudate extending upward from the lamina propria or by noninflammatory superficial exfoliation, it was concluded that the histopathologic findings did not support the contention that peptic esophagitis is caused by the corrosive action of regurgitated gastric juice.

Creamer [145] studied esophageal reflux by means of intraesophageal and intragastric pressure readings. Reflux occurred as a positive pressure wave in the esophagus on inspiration and was more marked in certain positions, indicating that reflux is present only during inspiration and changes in position may render the cardia incompetent. No appreciable change in the pressure across the cardia in various positions and no evidence of reverse peristalsis were observed.

By means of intraesophageal and intragastric pressure recordings Creamer [146] demonstrated that carminatives produce esophageal reflux similar to that in certain disease states.

Using the necropsy findings on 500 general hospital patients and 100 cases of sudden death, Lodge [147] found esophagitis in 36% of the hospital cases and 8% of the sudden death series. Reflux regurgitation of gastric contents appeared to be a factor in the tendency of esophagitis to occur in patients with severe vomiting or in whom prolonged gastric intubation was required. The effect of the horizontal posture and weakness of the diaphragmatic musculature in producing a similar reflux with resulting esophagitis in bedridden patients was stressed.

In discussing esophagitis and stricture from regurgitation Cranmer [148] pointed out that the diagnosis is established by the symptoms and confirmed by roentgen and esophagoscopic examinations. Vomiting from obstetrical and postoperative causes is one of the etiologic factors in the development of esophagitis. A description of the esophagoscopic findings was given in the five cases reported.

Taylor [149] discussed reflux esophagitis and the importance of esophagoscopy in the final diagnosis and evaluation of the disease. Esophagoscopy was advocated early so that medical management could be promptly instituted. While the commonest finding associated with reflux esophagitis was hiatal hernia differentiation from carcinoma was always necessary. Not only should esophagoscopy be performed in patients with roentgenologic evidence of esophagitis but persistent esophageal symptoms in the absence of positive roentgen findings should also require esophagoscopic study. The esophagoscopic findings varied with the stage of the disease, from acute esophagitis with a diffusely edematous and inflamed mucosa to actual ulceration with spasm of the musculature and later stricture formation. The usual finding was an incompetent cardia, and repeated esophagoscopy and biopsy was necessary in suspicion-arousing cases to differentiate carcinoma from the late stages of reflux esophagitis. In cases associated with hiatal hernia in which medical treatment failed surgical intervention was advised, as it was in those cases with ulcer or stricture where there was a history of bleeding, pain, and symptoms of perfora-

tion or obstruction unsuccessfully treated by bouginage.

Schmidt [150] discussed regurgitant esophageal ulceration on the basis of 170 patients with this lesion seen over a 10-year period. The majority occurred as complications of the sliding type of esophageal hiatal hernia or of operations upon the gastric cardia. Esophageal hiatal hernia exhibited an incompetent cardia with yellow-tinged gastric juice regurgitating into the lower part of the esophagus on esophagoscopy. Esophagogastrectomy was avoided in all benign lesions, for this completely abolished the sphincter mechanism of the gastric cardia. Dysphagia, retrosternal burning, epigastric pain, pyrosis, vomiting, and hemorrhage were the commonest symptoms. There were 38 patients with hematemesis and 20 with melena. Sixteen patients had bleeding sufficiently severe as to require transfusions. The symptoms of hiatal hernia were controlled by medical treatment, but when not, responsive repair of the hernia restored competency of the cardia and obviated the risk of the development of regurgitant esophagitis, ulcer, stricture, and shortened esophagus. In this series approximately 5% were treated surgically and the remainder were handled medically.

Sixty-six cases of reflux esophagitis were reviewed by Stewart and his co-workers,[151] and the outstanding finding was the presence of sliding hiatal hernia in forty-two patients. The common finding of hiatal hernia was demonstrated roentgenologically by proper positioning of the patient. Infrequent causes were gastric hypersecretion, postoperative states, protracted vomiting, pyloric obstruction, nasogastric intubation, ectopic gastric mucosa, and individual susceptibility. The majority of cases were treated successfully by a medical regimen with dilatations when needed. Surgical treatment was performed in appropriate cases before the esophagus became fibrotic and shortened. While impenetrable stenosis may indicate resection of a portion of the esophagus, a satisfactory operation and reconstruction has not as yet been developed.

Two cases of esophageal stricture in children caused by reflux esophagitis secondary to hiatal hernia and treated by surgical resection were described by Franklin and Henderson.[152]

In a discussion of peptic esophagitis and peptic ulcer of the esophagus Wolf et al.[153] described a third group, consisting of marginal esophagogastric ulceration. The ulcerative disease of the terminal esophagus was considered a distinct entity with special functional characteristics on the basis of the epithelium, which was either squamous or, oftener, heterotopic gastric mucosa. Discrete ulceration in the terminal segment was found in those cases regularly accompanied by regurgitation and a sliding hernia. The esophagoscopic findings were the presence of gastric rugae distal to the ulceration, regurgitation through the dilated channel, and obstruction requiring bouginage in many cases at the esophagogastric junction. After repeated esophagoscopies the ulcerations presented granular areas rather than whitish membranes.

Scott and Deutsch [154] reported seven cases of esophagitis associated with pregnancy. They were variable and consisted of simple erosive esophagitis, hemorrhagic esophagitis, stricture formation, and web formation, and in two cases a delayed stricture developed. Erosive esophagitis occurred in association with an otherwise normal pregnancy in women who vomited excessively, and in some it progressed to the serious complication of hemorrhage and esophageal stricture. The condition was occasionally encountered in pregnant patients in the absence of vomiting. The responsible factor seemed to be anatomic changes associated with pregnancy and responsible for gastroesophageal regurgitation with its resulting erosive esophagitis.

Thompson [155] recorded a case of peptic ulceration of the esophagus which perforated into the thoracic aorta.

Morris [156] described gastric epithelium lining the lower end of the esophagus and presented seven cases. The condition may be complicated by reflux esophagitis, high esophageal stricture, or by typical peptic ulcers, which may hemorrhage, perforate, or stenose on healing. Conservative medical management gave encouraging results.

Palmer [157] thought that the development of mucosal prolapse at the esophagogastric junction presupposed a point of mucosal fixation distal to the segment of potential prolapse. The condition was rare but was demonstrated by altering the thoracic-abdominal pressure through the Valsalva maneuver.

Esophageal Stricture

One hundred thirty-three cases of esophageal stricture following ingestion of sodium hydroxide (caustic soda) were reported by Marchand.[158] The severest strictures occurred in the lower esophagus, and solitary localized strictures were rare. The severest strictures were located in the retrocardiac position, but it was usual to find the upper esophagus also affected by stricture formation. The strictures were classified according to length and severity, and the esophagoscopic appearances of each were described. The system of grading proved useful in indicating the probable treatment needed for the particular case. In the Grade I strictures, i. e., those of mild severity, no active treatment was required, as they did not contract sufficiently to produce disability. The severer, Grade II, strictures required occasional dilatations, particularly during the early stages, but apart from the dangers of food impaction no further treatment was needed. The Grade III strictures, which were dense and thick although localized, always required treatment during the initial stages. Once an adequate lumen and dilatation had been accomplished the patient was able to maintain a satisfactory lumen by self-bouginage. Local operative treatment was seldom indicated, and even though considerable time was necessary conservative measures were persevered in Grade III strictures. The Grade IV strictures proved more refractory to conservative treatment, but attempts were made to dilate these conservatively. Once a stricture of more than several inches in length developed the epithelium could not cover the surface and conservative measures did not succeed, making surgery necessary. When the patient was not treated in the early stages the failure rate of dilatation was high, but even among those who were treated from the beginning 41% subsequently required major reconstructive surgery.

In a study of 97 patients with acute sodium hydroxide poisoning Marchand [159] reported the esophagoscopic findings in 94 patients, in whom the examination was performed within a week of injury. The 57 that were found to have esophageal corrosion were examined at weekly intervals, and in 16 of them healing of the corroded area without scarring occurred prior to the end of the second week. Forty-one patients developed strictures and five died, whereas fifty-one healed without serious esophageal involvement. The Salzer technique and corticotropin therapy proved ineffectual in preventing strictures, so that the present treatment consists of antibiotic control of infection and dilatations, which are started when the denuded areas become epithelized. With extensive severe strictures the prognosis was poor and dilatation rarely maintained an adequate permanent lumen, so that these patients developed complications throughout their lives.

Gregg and his associates [160] reported a case of lye ingestion in which the esophagus was not affected, while the buccal pharyngeal mucosa and stomach lining were extensively destroyed. This unusual finding was explained theoretically on the basis of short contact of the caustic solution with the esophageal mucosa, rapid passage into the stomach, the buffering effect of milk, mucous blanket covering the esophagus protecting the epithelium, and neutralization of the alkali in the stomach by the gastric

acid, preventing burns when the solution was vomited.

Four cases with stenosis of both esophagus and stomach following the ingestion of sodium hydroxide in three and hydrochloric acid in one were discussed by McKibben and Lee.[161] Although stenosis of the esophagus was readily treated by dilatation, it was necessary to employ surgical treatment for the lesions involving the stomach, particularly the pyloric area.

Two cases of ammonia corrosion of the esophagus verified by esophagoscopy were treated solely with cortisone and antibiotics by Lund-Iversen[162] with favorable results. No complications developed, the esophagus healed without scarring, and the esophagoscopic findings were normal.

Flexible spring or rubber to replace string guides in the conservative treatment of stenosing diseases of the esophagus, both benign and malignant, was preferred by Puestow.[163] When this method was used in combination with radiation therapy a patient with squamous-cell carcinoma of the upper end of the esophagus lived 11 years after the onset of symptoms.

Lawrence and Burford[164] advocated esophageal resection and esophagogastric anastomosis in the treatment of esophageal caustic burns with stricture formation. Both experimental and clinicopathologic findings indicated the necessity for placing the esophagus at rest during the acute phase of injury. Dilatations of established strictures were considered hazardous and the results disappointing due to fibroplasia and abscess formation.

Intraluminal resection of an esophageal stricture using a specially designed ribbon esophagoscope and esophageal resection knife was carried out by Emerson Jr.[165] The stricture was resected, removing a wedge-shaped area of mucous membrane and fibrous tissue, and was repeated at three different intervals. Short-time follow-up of the case reported indicated a satisfactory lumen for swallowing.

Diverticulum of Esophagus

A case of esophageal obstruction produced by five traction diverticula which responded to conservative treatment was presented by Slanger.[166]

Benign Lower Esophageal Lesions

In discussing benign lesions of the lower esophagus with special reference to hiatal hernia, epiphrenic diverticulum, and cardiospasm Putney[167] pointed out that in some cases it is necessary to render treatment with a view toward relieving symptoms rather than obtaining complete cure. In any surgical treatment disruption of the hiatal pinch-cock results in reflux esophagitis with regurgitation and pain, and when patients are treated surgically this mechanism should be kept intact. The symptoms of benign lower esophageal diseases were similar, and the diagnosis depended upon thorough roentgenologic and esophagoscopic study. The chief difficulty in diagnosis occurred when two or more conditions were present, particularly those affecting other portions of the gastrointestinal tract, and the cardiovascular or respiratory system.

Boyd[168] found that only a small minority of 500 patients with hiatal hernia and cardiospasm had esophagitis. For the simple lesions that represented the majority of the cases medical measures alone or combined with periodic dilatations afforded comfort and good nutrition. Medical measures with periodic dilatations sufficed for some of the patients with esophagitis and cardiospasm. Other patients with hiatal hernia, marked esophagitis, and regurgitation were greatly improved by repair of the hernia.

Routine investigation of 170 adult patients with hiatus hernia by Palmer[169] revealed that 15% also had gall stones and diverticulosis of the colon (Saint's triad). The association appeared so frequently that when a hiatus hernia patient had either gall stones or diverticulosis the third disease was suspected. Since the results of cholecystectomy were uniformly poor, the hiatal hernia

was found to be the factor responsible for the symptoms.

Cardioesophageal Relaxation

The term cardioesophageal syndrome was used by Forshall [170] to cover all cases of incompetence of the esophageal hiatus in childhood. In the last three and one-half years 93 instances were encountered, and in 58 cases the incompetent cardia was located below the diaphragm and was designated as lax esophagus. The esophagoscopic findings in this group included marked dilatation of the lumen with constant regurgitation of gastric contents, patulous hiatus without constriction, and various degrees of esophagitis involving the lower third of the esophagus. Medical treatment consisting of small, frequent feedings in the upright position, thickening of the feeding mixture, and antacids proved highly successful in this group. When the incompetent hiatus was situated above the diaphragm the lesion was termed a sliding hiatal hernia, and this was demonstrated in the remaining 35 children. The esophagoscopic findings were identical with those of the lax esophagus group, although the acuteness of the esophagitis had a tendency to vary more in the infants with hiatal hernia. Surgical treatment was advocated for sliding hernias in children, since medical treatment failed in all instances and the incompetent hiatus exhibited no tendency to become normal. The nature of the factors responsible for cardioesophageal relaxation has not been determined, but some evidence pointed to a distinct and separate etiology of lax esophagus and sliding hiatal hernia.

Hiatus Hernia

In discussing the symptoms of hiatus hernia Leather [171] concluded that pain simulating that of myocardial ischemia was a rare feature of hiatus hernia. In 54 of 70 consecutive cases in which hiatus hernia was the only detectable lesion the symptoms were epigastric pain, heartburn, acid regurgitation, flatulence, and attacks of vomiting, all of which were aggravated by postural changes.

The problem of partial thoracic stomach and esophageal hiatus hernia in infancy and childhood was thoroughly discussed by Swyer.[172] Two groups of cases were recognized, one due to developmental shortening of the esophagus and the other due to herniation of the stomach through the esophageal hiatus. Contrary to popular belief, the acquired hernias through the hiatus far outnumbered the congenital cases. The hazard of hiatus hernia in children was incompetence of the esophagogastric pinchcock with resulting esophagitis, malnutrition, and stricture formation. Esophagoscopy proved indispensable in detecting esophagitis and following its response to treatment. The management was directed at preventing gastroesophageal reflux by maintenance of a nearly upright position throughout the 24 hours, reinforced by thickened feedings and antacids. Paraesophageal hernia in childhood was considered an absolute indication for operation, and repair of the hernia was advised in other esophageal hiatus hernias not controlled by conservative measures.

In a study of 58 cases of hiatus hernia in children, 14 esophagoscopies in 9 patients were performed by Thomsen.[173] The esophagoscopic findings were esophagitis in all patients and strictures in some. In two patients a foreign body immediately above the stricture was removed. Biopsy above the stricture was performed in one case, and the histologic report was esophageal mucosa with fibrosis.

In a review of 70 consecutive cases of hiatal hernia Hall and Newton [174] described the esophagoscopic findings as classical. In more than half of the series roentgen examination failed to demonstrate the hernia. Esophagoscopy was found diagnostic in the absence of roentgenologic evidence of the disease, determined the presence and degree of esophagitis, uncovered any stricture, and allowed dilatation and biopsy of the stric-

ture. Operation in the presence of esophagitis was advised, while in its absence surgery was safely deferred.

Blades [175] brought out that endoscopic examination of the esophagus and upper stomach in hiatal hernia has not been performed frequently enough. Endoscopic examination was particularly necessary to differentiate between acquired short esophagus and congenital short esophagus by the appearance of the esophagogastric junction. In the congenital type the esophagoscopic junction was open and the esophagoscope entered the stomach with ease, while in the acquired type resistance was encountered. This distinction was important to the surgeon considering surgical treatment. Another reason for endoscopic examination was the occasional finding of carcinoma as the underlying factor in the production of the hernia.

In his vast experience with esophageal hiatal hernia Harrington [176] considered the condition primarily a mechanical one and employed surgical treatment in a series of 489 cases. Esophagoscopic examination was essential to determine the possible presence of lesions associated with the hernia, such as esophagitis from reflux gastric secretions, peptic ulceration of the esophageal mucosa, traumatic ulceration at the cardia, stricture, and spasm of the esophagus. Esophagoscopy was also important in determining the possible presence of conditions not related to the hernia, such as carcinoma in the lower esophagus or cardia, mucosal or intramural tumors or cysts, and diverticula of the esophagus. The findings of lesions complicating the hernia as well as lesions not related to the hernia often changed the method of management as well as the type of treatment employed.

Smithers [177] considered the association of cancer of the esophagus and stomach with sliding hiatal herniation too common to be due to chance and reported 12 cases to substantiate his belief. Reflux esophagitis and ulceration associated with sliding hiatal hernia may predispose to the development of esophageal tumors but seems unlikely to be the major factor involved. The occurrence of adenocarcinoma of the esophagus associated with hiatal hernia is due to many factors, two common ones being the likely spread in the submucous lymphatics of the esophagus by tumors of the cardia and the frequent lining of a small portion of the lower esophagus by glandular epithelium, which is liable to malignant disease at the constricted area.

Smith [178] reported a case of carcinoma developing in a thoracic stomach. It was known that the stomach had been herniated into the thorax for at least 10 years, and the carcinoma developed in the body of the stomach not associated with the esophagogastric junction.

Esophageal Varices

Kirsh and his associates [179] examined the esophagus of 502 patients with diseases of the liver and spleen for evidence of varicosities both by roentgen and esophagoscopic studies. In this group varicosities were shown by both roentgenograms and esophagoscopy in 66 cases, while in 388 patients no varicosities could be demonstrated by either method. Of the 102 patients with positive findings on esophagoscopy 66 had positive findings on roentgen examination.

Brick and Palmer [180] reported the results of esophagoscopy in the detection of esophageal varices in 172 patients with cirrhosis of the liver. A diagnosis of esophageal varices was made by esophagoscopy in 108 patients, while in only 24 of these patients were varices diagnosed on the roentgenogram. Of the 64 patients with a history of hemorrhage, varices were diagnosed by esophagoscopy in 51 and by roentgen examination in 19. In the 108 patients without a history of bleeding, esophagoscopy was positive in 57 and in only 5 were the roentgenograms positive. The more frequent finding of varices after hemorrhage was attributed to the fact that the varices were more apt to be larger in patients with hemorrhage than in those without bleeding.

In this study roentgen diagnosis was reliable in only 22% of the cases found by esophagoscopic examination.

The esophagoscopic findings on 674 patients with liver disease with particular emphasis on esophageal varices were reported by Friedberg et al.[181] Of the 582 patients with hepatic cirrhosis, esophageal varices were diagnosed endoscopically in 175 patients, or 30%. When there was a history of melena, hematemesis, or both the finding of varices on esophagoscopy rose to 49%. While the diagnosis of esophageal varices was made oftener endoscopically than by roentgen examination, corroboration of the diagnosis by both endoscopy and roentgenography was found in over 80% of the total patients studied. No significant hazards of bleeding during esophagoscopy were encountered. Variations in portal hypertension were indicated by the fact that in approximately 10% of the patients varices were present at one examination and absent at the following ones. Spontaneous disappearance or at least transient esophageal varicosities were established by these findings.

Brick et al.[182] reported an esophagoscopic diagnosis of varices in 161 out of a total of 238 patients with cirrhosis, and in 87 of the positive cases there was no history of hemorrhage. Of 147 cases studied both esophagoscopically and roentgenologically, 92 patients were diagnosed as having varices by esophagoscopy and only 19 by roentgen study.

Palmer and Brick [183] found varices of the distal esophagus in the absence of portal and superior vena caval hypertension in 3.7% of a group of 350 patients with esophageal varices studied by esophagoscopic methods. While the diagnosis was difficult, it could be established by measuring the venous pressure directly within the varices; otherwise, the diagnosis was one of exclusion. Esophageal varices without venous hypertension may be responsible for hemorrhage, but it seemed more likely that esophagitis was the causative factor.

By esophagoscopic observations Palmer and Brick [184] demonstrated that esophageal varices occurred in liver disease other than cirrhosis. Many clinical situations not previously suspected of being accompanied by portal hypertension, such as chronic congestive heart failure and the latter half of pregnancy, caused varices, although they rarely produced significant hemorrhage.

Macbeth [185] considered the injection of sclerosing solutions in the esophageal varices of portal hypertension a safe and satisfactory alternative to major surgery, as evidenced by experience in 14 cases with liver damage (intrinsic) and 16 cases without liver disease (extrinsic). Treatment was directed toward preventing hemorrhage and was only performed on those veins prolapsing into the esophageal lumen. The sclerosing injections controlled bleeding indefinitely in the extrinsic cases and for limited periods in the intrinsic cases. Splenectomy made the veins more easily controllable and was carried out as an adjunct to injection therapy. Esophagoscopic treatment was done once or twice a year as new varices formed. The sclerosing solution was sodium morrhuate injection (5%), and 3-4 ml. was injected at each puncture site. No harm resulted from six injections of this size at one session. As large an esophagoscope as possible was used to place the veins upon a stretch and to obliterate the vein after injection by passing the instrument beyond the point of injection. If the patient had recently bled the varices nearest the cardia received the first injections, but in a quiescent phase it was more convenient to treat the veins in groups from above downward. A gauze pack was held over the site of venepuncture to control oozing when necessary, and if bleeding was marked a hydrostatic bag was used for its control. The initial treatment was repeated every one to two weeks until no visible veins remained uninjected. Following injection therapy substernal pain was usual but slight. Ulceration was an occasional complication, while venous embolism and

allergy to the injection fluid were not encountered.

Cardiospasm

Holinger and his associates [186] reviewed 189 cases of cardiospasm and correlated the etiologic, medical, endoscopic, and surgical phases of the problem. Although the etiology has not been definitely determined, most of the evidence points to neuromuscular dysfunction of the entire smooth muscle portion of the esophagus, due primarily to disease of the myenteric plexus. In this series there were 19 cases with obvious psychoneurosis or significant psychogenic factors initiating the cardiospasm, although no detailed study was made of the patients in this regard. The outstanding symptom was dysphagia, and when pain was present it usually signified an appreciable degree of esophagitis. Weight loss was frequently marked, and pulmonary symptoms due to aspiration of food contents occurred frequently. The distinguishing point in the history of patients with cardiospasm was fluctuation of symptoms for a considerable period of time, often influenced by emotional reactions, whereas the symptoms of patients with carcinoma progressed steadily. In addition, the patients with cardiospasm usually had symptoms of longer duration than those with carcinoma. Esophagoscopic studies were considered a routine and necessary diagnostic step in establishing the presence of cardiospasm as well as in controlling therapy. Esophagoscopy served not only to confirm the diagnosis and rule out carcinoma but allowed the mucosal changes to be inspected before the institution of treatment. In uncomplicated cardiospasm the esophageal picture revealed retained secretions and undigested food together with various degrees of esophagitis and dilatation of the lumen. Unless there was angulation and the case was one of long standing, the cardia could be visualized and passage of the esophagoscope into the stomach was unimpaired. In treatment drug therapy was beneficial and symptomatic relief was obtained by various medical means. Active treatment consisted of esophageal dilatation and external surgical procedures. The response to dilatation was satisfactory, but two perforations occurred while the patient was undergoing pneumatic bag dilatation from too forceful stretching of the musculature and a resulting mucosal tear. In the surgical treatment the Heller muscle-splitting operation was preferred, but more extensive procedures, including gastroesophageal anastomosis without resection of the cardia, were required for the more advanced and extensive cases in which the walls were markedly atonic. Postsurgical treatment presented a problem, for the 18 patients who had from one to three operations on the esophagogastric junction 13 required dilatation following the surgery. Accurate results of therapy were not determined for at least three to five years because relief followed almost any type of active dilatation or surgical correction for the first few years but the recurrence rate was high.

Benign Tumors of Esophagus

Puestow et al.[187] reported 12 cases of benign esophageal tumors, 1 of which was a cavernous hemangioma. On esophagoscopic examination of this case a nonobstructive submucosal mass was seen just below the cricopharyngeus. The mass could be easily compressed with the esophagoscope and resumed its shape on withdrawal of the tube. The tissue removed for histologic study was not deep enough to secure the lesion, and the diagnosis was made at operation.

LeJeune [188] described a benign pedunculated esophageal tumor in which death occurred following a bout of coughing when the tumor was regurgitated from the esophagus and became lodged in the aperture of the larynx, causing asphyxia. The tumor was pedunculated, allowing for its easy movability, and on esophagoscopic examination the tumor was hard and fixed in position, so that no pedicle was discernible. While esophagoscopy was useful in diagnosis it did not help in treatment, and at-

tempt at removal with a wire snare was unsuccessful. Repeated attempts at biopsy were necessary to secure tissue for histologic examination.

A history of regurgitation of a tumor mass preceded the diagnosis of a benign fibromatous polyp of the esophagus, as described by Lodmell.[189] On esophagoscopy the entire esophagus was filled with a smooth tumor whose mucosa appeared intact. The site of origin could not be determined until operation, when it was found attached near the cricopharyngeus muscle.

Two cases of leiomyoma of the lower third of the esophagus were reported by Piacentini.[190] In the one case in which esophagoscopy was performed the lower third of the esophagus was dilated and immediately above the cardia two roughly rounded pendulous lesions which were completely covered by mucus were observed. These growths were firm in consistency, not ulcerated, and did not cause obstruction to the food passage.

Kinnear[191] presented a case of lipoma of the esophagus which was not pedunculated but was bulky and situated submucosally in the lower half of the esophagus.

Esophageal Cytology

Johnson et al.[192] reported the results of their studies on esophageal washings of 364 patients with malignant and benign diseases of the esophagus. Of the 148 patients with malignant tumors primary in the esophagus, cytologic examination yielded a positive diagnosis in 69% and was suspicious in 12.2%. Negative reports were obtained in 18.2%. The failure in most cases to make a definite diagnosis of cancer was due to the absence or scarcity of tumor cells in the specimens. Of 81 patients with malignant tumors primary in other organs, mainly in the stomach, a positive diagnosis of cancer was obtained in 25, whereas in 44 the report was negative or insufficient for diagnosis, and in 12 suspicion was noted. In the 135 patients in whom no malignant tumor was demonstrated a positive diagnosis was made cytologically in 3 and a report of suspicion was made in 7. In 57.4% of the patients with malignant tumor primary in the esophagus both cytologic examination and biopsy yielded a positive diagnosis. In 21.7% biopsy was positive and cytologic examination was negative or inconclusive. In 12.2% cytologic examination yielded a positive diagnosis of cancer, while biopsy was either negative or not done. In 8.8% neither method established the diagnosis. While cytologic examination of esophageal washings did not replace biopsy and added only a few positives to those obtained by biopsy, it proved of value in cases in which biopsy was not practical. In the cytologic study the most important source of diagnostic error was in the cytology of severe esophagitis of long standing.

Klayman[193] examined 40 patients in whom pathological lesions of the esophagus were suspected and performed cytologic examination by lavage with isotonic solution of Ringer's solution using a Lævin tube. Malignant growths were present in 20 patients, and esophagoscopic examinations revealed the malignant lesion in 15 with positive biopsy in 12. In 19 of the 20 patients a positive diagnosis of malignancy was made on cytologic examination, and there were no false positive reports. In the 20 patients with benign lesions the cytologic examinations were all correct. Two patients with cardiospasm developed malignancy years later after extensive treatment by dilatation, and the cancer was detected by cytologic examination. In one case with unexplained jaundice, no esophageal symptoms, and negative roentgenograms malignant cells were recovered from an unanticipated carcinoma of the esophagus.

Malignancy of Esophagus

A case of intramucosal esophageal carcinoma with involvement of almost the entire esophageal mucosa without a localized mass and with only limited superficial invasion of the submucosa and muscularis was described by O'Gara and Horn.[194] No symp-

toms referable to the esophagus were present during life, and the presenting complaint was a lump in the neck due to cervical lymph node metastasis.

Dysphagia in carcinoma of the esophagus may be a late symptom, as emphasized by Wohl et al.[195] Unusual presenting symptoms occur as a result of extension to adjacent structures or from distant metastasis. Hoarseness, cough, anorexia, epigastric pain, and mass in the neck were observed as initial symptoms, with dysphagia developing later.

Palmer[196] listed 13 out of 100 cases of esophageal carcinoma in which the first biopsy failed to secure tumor tissue. The causes for failure were the intramural position of some infiltrating tumors, obstruction which prevented access to ulcerated tumor tissue, and simple misdirection of the forceps.

A case of primary melanocarcinoma of the esophagus was reported by Pomeranz and Garlock,[197] who believed that the disease arose not by in situ transformation of squamous mucosa but from melanoblasts ectopically located in the esophagus.

Puncture of the cervical esophagus by means of a trocar and cannula in the palliative treatment of carcinoma of the esophagus was described by Davidson.[198] Through the cannula either a slender polyethylene (Polythene) tube or a thread to which a tube was anchored was passed through the malignant stricture. The passage of the trocar and cannula was accomplished under antibiotic cover and with general anesthesia. After passage of the esophagoscope (Negus type) to the level of the suprasternal notch, it was rotated so that the oblique lower opening pointed forward and to the left toward the anterior border of the sternomastoid muscle. Withdrawal of the instrument was begun until the operator felt its lower end through the tissues of the neck at the anterior border of the sternomastoid muscle just above the left clavicle. The head was not extended, so that the neck was loose. A needle was then passed through the skin at the anterior border of the sternomastoid muscle into the open lower end of the esophagoscope, where it was visualized through the tube. After confirmation that the needle had been properly placed it was withdrawn and a trocar and cannula, 10 cm. long and 3 mm. in diameter, were inserted in the same manner. The only anatomic structure requiring special attention was the trachea, and the needle was kept close to the left side of the trachea. In passing the polyethylene tube the distal end was first threaded through the stricture via the esophagoscope guided by side-curved forceps, and then the proximal end was brought out through the neck after introduction of a flexible dilator from the neck into the esophagoscope and attachment of polyethylene tube to it. A plastic tube may be inserted through the stricture via the esophagoscope and a thread attached to its upper end brought out of the neck in the same manner. The thread was tied over a button, thus preventing the tube from slipping downward. This method was employed in nine cases without immediate or late complications and was effective over a period of time in allowing the passage of saliva and food with minimal distress to the patient.

Coyas[199] employed a plastic tube for feeding purposes as a palliative measure in carcinoma of the esophagus. After the introduction of a large-lumen esophagoscope the malignant stenosis was explored with a bougie to locate the remaining lumen. The plastic tube was placed through the obstruction by means of a bougie, to which it had been attached. After withdrawal of the bougie the position of the tube was verified by gentle aspiration. Intubation was possible in 12 patients, in 1 of whom perforation and an esophagotracheal fistula developed on the second day, in 2 the tube slipped out of place and was passed by rectum, and in 2 others obstruction took place because of impaction of food and regrowth of the tumor. The advantages claimed for this method of intubation were as follows: The tube is light in weight; has a conical end,

facilitating placement and lessening the risk of trauma; is malleable, molding itself better to the lumen, and the bougie is graduated and incorporated with the tube, making positioning more accurate.

Berg [200] described 12 cases of esophageal herpes occurring terminally in cancer patients. The common factor was esophageal trauma by irradiation or surgery. The clinical importance of herpetic esophagitis was not determined, but it possibly could lead to infection, ulceration, and necrosis. The practical importance of the lesion was its possible confusion with carcinoma in situ.

Sarcoma of Esophagus

Bell [201] described a case of fibrosarcoma of the middle third of the esophagus treated by deep x-ray therapy who was alive and well 2½ years later. On esophagoscopy a large friable growth was found 26 cm. from the upper teeth and a large piece of tissue was removed for histologic study. The diagonsis of fibrosarcoma of the esophagus was made. Exploratory thoracotomy was performed, but the lesion was considered inoperable because of extension into the hilum of the left lung and irradiation therapy was given.

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Abstracts from Current Literature

Ear

A New Technique for Bone Conduction Testing. David Higbee, Ann. Otol. Rhin. & Laryng. 65:29 (March) 1956.

Higbee, in studying bone-conduction testing, has placed the bone-conduction vibrator on the forehead, held by constant pressure through the use of an inflatable rubber belt and manometer inflated to 40 mm. Hg.

Selective masking consists of sawtooth noise fed through the headphone after the noise is adjusted for each frequency to a level allowing the pure tone to be heard at the threshold in the ear under test.

It was found, in testing 100 ears for thresholds at 500, 1000, and 2000 cps, that an average of 10 db. more sound pressure was required when the vibrator was placed on the forehead than on the mastoid. Therefore, the bone readings were elevated 10 db. for each frequency when findings were recorded on the audiogram.

The above methods of more accurate placement of the vibrator and constant pressure at point of contact with the cranium should make conduction readings more reliable.

Steele, Kansas City, Kan.

Hearing Impairment in Noisy Environment. J. Coleman Scal, New York J. Med. 56:2839 (Sept. 15) 1956.

Industrial workers may become deafened by noises due to their occupation. This is of great concern to the worker, his employer, insurance companies, workmen's compensation bureaus, and to industry in general. Such deafness begins slowly, and the victim may not be aware of it until tinnitus becomes troublesome. The high tones seem first to be affected, a loss at the 4000 frequency. When he notices that he cannot hear the doorbell or telephone, this is probably due to perceptive deafness. Later the lower tones are involved with loss for near conversation, such as at the table. He also has to increase the loudness of the radio and television. "Noise is unwanted sound." It also can be described as "waves of compression and rarification transmitted through the air." The first complaints may be tinnitus and temporary loss of hearing, which disappear after the worker goes off the job. This is nerve fatigue. If it continues, the acoustic trauma worsens and may become permanent. The audiometer is useful, but is subject to variations in graphs taken by different machines and by different persons. The examiner's own mental and physical status must be compared with that of the examinee! "Fowler's recruitment test is . . . used for differentiation of perceptive deafness from conductive deafness." In examining older persons, thought must be given to senile atrophy of the eighth nerve, but presbyacusis may be present in which sounds may be painful owing to hypersensitivity. When the noise level of a work area is above 90 db., acoustic trauma is likely and inattentive deafness may supervene. The faculty of listening is dulled or lost.

Vascular disturbances account for tinnitus. The internal auditory artery does not supply sufficient blood to the end-organs. Therefore, venous stasis and arterial compression are possible. They may be associated with Ménière's disease. Fowler Jr. speaks of a "sludging of the blood" in the terminal arteries.

Deafness resulting from constant trauma becomes cumulative over the years. It is permanent. However, total deafness has not been reported, that is, a hearing loss of 80 db., which is complete deafness for all ordinary communications. Hearing should be tested at the beginning of employment, and retested at least every six months. There is, of course, no treatment save change of occupation. Curiously enough, a few workers seem unsusceptible to these pernicious noises and remain unaffected, even after 10 years' employment.

Abstracter's Comment: Residual deafness, reduced hearing, is common when pathology of the drum and middle ear persists. Therefore, it is paramount that a careful reading of hearing ability be taken at the very beginning of employment. And Dr. Scal's insistence upon repeated tests is sound advice. One infers that he thinks deterioration may cease upon

change of environment to quieter surroundings. That is problematical, and considered opinion of other authorities seems to lean to progressive deterioration, slowly but without let-up. In any case there is little that the otologist can do through treatment or drugs to avert the end-result. Prevention is the watchword!

I. W. Voorhees, New York.

The Investigation of Vestibular Function. Terence Cawthorne, M. R. Dix, C. S. Hallpike, and J. D. Hood, Brit. M. Bull. 12:131 (May) 1956.

All these researchers have appointments in the National Hospital, Queen Square, London, and with various "Councils" for investigation of medical subjects. The subject of vestibular function occupies 12 pages, and contains 12 figures and 40 references to the literature. The general outline presented is as follows: *A*, introduction; *B*, caloric tests; *C*, rotational tests; *D*, positional tests of otolith function; *E*, tests for optokinetic nystagmus. "For the introduction of these tests we are indebted, for the most part, to Bárány. For his work upon them he was awarded the Nobel Prize in Medicine in 1914, and they have since come to constitute the chief stand-by of all who are called upon to undertake the objective examination of vestibular function." Although there have been some objectors to Bárány's methods and conclusions, "Bárány's concept may be said to have maintained its position without serious challenge." In 1942, Fitzgerald and Hallpike described a new caloric test procedure which fulfills the following requirements: (1) the tests should be easy to apply and not uncomfortable to the patient, (2) the results should provide an accurate and repeatable measure of the sensitivity of the external canal, and (3) they should be capable of revealing the phenomenon of directional preponderance. Then follow figures and diagrams to illustrate this new procedure. The same in general may be said of the description of the rotational tests. For the positional tests of otolithic function, Dix and Hallpike speak of the "benign paroxysmal type of positional nystagmus." This was known to Bárány also, and was emphasized by Nylén, pupil and successor of Bárány at Uppsala University. One of the illustrations of the text shows the Bárány drum used to test optokinetic nystagmus. These latter tests are of special value in the diagnosis of cerebral lesions, such as intramedullary neoplasms and disseminated sclerosis.

Abstracter's Comment: It is heartwarming to read of the firm place in otologic science held by Bárány so many years after his untimely death, in America. He was one of the great pioneers in this field, and his industry knew no bounds. He cared nothing for private practice, and was always to be found in his laboratory. I take pride in being the first to translate Bárány's "Funktionelle Prüfung des Vestibularapparates," which was published in our American *Annals of Otology, Rhinology and Laryngology* in 1912. It was a labor of love, but the most arduous task, because of technicalities, which I have ever known.

I. W. Voorhees, New York.

Pathology, Symptomatology and Diagnosis of Organic Affections of the Eighth Nerve System. E. A. Carmichael, M. R. Dix, and C. S. Hallpike, Brit. M. Bull. 12:146 (May) 1956.

There are four segments to this study: the labyrinth, the eighth nerve, connections of this nerve to the brain stem, and associated disorders of the cerebral hemispheres. As for the labyrinth, both the auditory and cochlear elements are ordinarily affected together, whatever the cause. The otologist is concerned with Ménière's disease, and positional nystagmus of the benign paroxysmal type. Ménière made an exhaustive and accurate study of a disorder of middle life which occurs "regardless of sex." It does not shorten life, and is not associated with any other organic disease, "including allergy, outside the eighth nerve system." Basic symptoms are deafness and tinnitus, with paroxysms of vertigo, vomiting, and nausea superimposed. There is distorted perception of speech and music. Perceptive deafness is always present with loudness recruitment and often "overrecruitment." Hearing aids accomplish little or nothing. Vestibular reactions are commonly abnormal, and nystagmus is present during attacks, although absent between them. Caloric tests always bring forth abnormalities in some 90% of all cases examined.

Eighth nerve neoplasms develop insidiously with unilateral deafness, rarely accompanied by severe, overwhelming tinnitus. Both functions of the eighth nerve may be virtually destroyed by tumors without urgent complaints from the patient other than unsteadiness of

momentary duration. Otological signs and symptoms are rather constant, comprising abnormal caloric responses on the diseased side and spontaneous nystagmus.

Perceptive deafness of any degree arises from destruction of cochlear nerve fibers and hair-cell degeneration. Loudness recruitment, so typical of Ménière's disease, is absent or incomplete. Changes in the cochlear blood supply may vitiate the above findings. Nearly all eighth nerve tumors pass through the otological stage and may be readily diagnosed. "Removal of the tumors in this stage is easy, and recovery without neurological disability rapid and uneventful . . . useful cochlear function may sometimes be preserved."

Connections of the eighth nerve with brain areas have been worked out by pathologists, but are not yet quite complete. Vascular lesions of the brain stem, particularly those of the posterior-inferior cerebellar artery, have been established as to other cranial nerves than the eighth. Points of emergence of the cranial nerves are usually well below that of the eighth; the cochlear fibers go to join the trapezoid body, and travel upward, mainly upon the opposite side of the brain stem. "By contrast with the vestibular elements, there are no cochlear elements below the level of entry of the eighth nerve." Table II consists of a chart showing symptoms and signs of organic disease of the eighth nerve system. Obviously it cannot be reproduced here. Organic affections of the eighth nerve system are shown in Plate III associated with an explanation of the text. These plates are all excellent in detail and resemble engravings.

I. W. Voorhees, New York.

Artificial Tympanum Made of Yougovinyl. P. Zdravev, Ann. oto-laryng. 73:283 (April-May) 1956.

The author uses a prosthesis of a plastic material to close the perforations in the tympanum. He shapes the prosthesis according to the size and shape of the perforation and applies it to the tympanic membrane. This can be directly applied to the perforation. The indications for its use are noted, and the prosthesis is recommended for patients who have less than a 40 to 50 db. hearing loss. Patients who have greater losses require some further measures, such as rehabilitation, surgery, or an electric prosthesis.

Persky, Philadelphia.

Artificial Hibernation in the Treatment of Otitic Abscess of the Cerebellum. Flottes, Picard, Devilla, de la Bernardie, and Navarrone, J. franç. oto-rhino-laryng. 4:527 (Oct.) 1955.

The authors present an extremely interesting case of cerebellar abscess which was treated with the usual antibiotics and surgical drainage; but, because of the progression of symptoms, they decided to institute artificial hibernation. After this was instituted and maintained for a period of time, the patient showed a remarkable improvement in his condition and was finally discharged from the hospital as cured. However, a few residual symptoms remained, i. e., a retarded mentality and a slight diminution in muscular ability of the left leg. In conclusion the authors believe that artificial hibernation is an extremely important adjunct in the treatment of cerebral infections in that it can slow down the vital functions of the body, thus not only preventing the spread of the infectious process but also enabling both medical and surgical procedures to effect a proper elimination of the infectious process.

Persky, Philadelphia.

Is Streptomycin Sulfate Toxic for Audition? A. Appaix and J. Henin, J. franç. oto-rhino-laryng. 5:135 (March-April) 1956.

The authors have reviewed their experiences with both streptomycin sulfate and dihydrostreptomycin in their action on the cochlea. They quoted the results published by many authors in this so-called relationship to the vestibule and the cochlea. They have studied 150 patients who have received varying amounts of streptomycin ranging from 30 gm. to more than 300 gm. They have divided this group of tuberculous patients into five categories according to the amount of streptomycin that they had received. In this series 53 patients had normal audiograms, almost equally divided among the five groups. The remainder have various forms of deafness, which may have been present prior to the initiation of streptomycin therapy. The authors state that they have never observed any modification in the audiometric curve in any of the

patients who received less than 300 gm. of streptomycin. Two factors seem to influence the problem of streptomycin intoxication: One is allergy, and the second is the faulty renal elimination of the antibiotic, thus causing an abnormally high concentration in the blood. It is their feeling that in a number of patients where deafness has ensued there is a possibility of their having had an associated tuberculous meningitis. On the same basis, it is felt that the injection of the antibiotic into the cerebral spinal fluid acts more directly on the hearing loss than the intramuscular route. They present a series of 20 patients who have been studied, some of whom have had normal hearing difficulty prior to the initiation of antibiotic therapy. None of these patients showed any appreciable change in their hearing acuity following streptomycin therapy. In consequence, the authors have raised the question "Does streptomycin actually exert any influence on the cochlear apparatus, or is there some associated tuberculous meningitis which is the actual cause of pathology?" They believe a further careful evaluation of both the intracranial pathology and the effect of the antibiotic should be made.

PERSKY, Philadelphia.

EMPYEMA OF THE ENDOLYMPHATIC SAC. P. ZDRAVEV, Rev. laryng. 77:508 (May-June) 1956.

The author presents two patients with abscess in the endolymphatic fossa. The first case was that of an otitis media complicated by mastoiditis, facial paralysis, meningitis, and cerebellar abscess. Intracranial surgery was performed, draining the cerebellar abscess, but the progress of the patient was unsatisfactory and became critical. The patient was reoperated on. The endolymphatic fossa was entered by the Portmann approach, and an abscess was found in this area. This was drained, and the patient made a slow but uneventful recovery. The second case was that of a mastoiditis, cerebellar abscess, and lateral sinus thrombosis. Here the lateral sinus was opened, the jugular vein was ligated, the cerebellar abscess was drained, and on exploration of the endolymphatic fossa an abscess was found. This was opened and drained. This patient also made an uneventful recovery. The purpose of this presentation is to draw attention to this area, in the presence of intracranial complications. Fortunately, because of the antibiotics, empyema of this area is relatively rare. On the other hand, when present and unsuccessfully treated or unrecognized, empyema may lead to fatal results.

PERSKY, Philadelphia.

Larynx

THE CLINICAL SIGNIFICANCE OF THE VALLECULA. FREDERICK T. HILL, Ann. Otol. Rhin. & Laryng. 65:68 (March) 1956.

Hill makes a plea for the routine and systematic use of the laryngeal mirror for inspection of the vallecula, which frequently contains much of interest to the otolaryngologist.

Clinical entities of significance are hypertrophy of the lingual tonsils, acute lingual tonsillitis, abscess of the lingual tonsil, keratosis, cysts, varicosities, and foreign body. More rarely seen diseases are carcinoma of the lingual tonsil and the presence of lingual thyroid.

STEELE, Kansas City, Kan.

THE SURGERY OF CANCER OF THE LARYNX. JOHN J. O'KEEFE, Ann. Otol. Rhin. & Laryng. 65:131 (March) 1956.

O'Keefe sets up five criteria to consider in the surgical treatment of laryngeal cancer: (1) location and extent of the growth, (2) presence or absence of impairment of motility, (3) presence or absence of metastasis (local or remote), (4) grading according to Broder's classification, and (5) general condition of the patient.

Failure to appraise the patient with these criteria in mind will result in failure of the entire effort.

Laryngofissure is an effective surgical method for the resection of malignant lesions entirely contained on the vocal cord. When local laryngeal recurrences follow the procedure, they are usually directly attributable to improper selection of thyrotomy or attempted extention of the operation to accommodate lesions which extend beyond the confines of one vocal cord. The intralaryngeal resection of small malignant lesions,

entirely limited to one vocal cord, by suspension laryngoscopy as reported by Lynch, LeJeune, New, and others is not mentioned by the author.

Laryngectomy is reserved for the resection of malignancies contained within the larynx and should be associated with radical neck resection oftener. This technique is positively indicated when cervical nodes are palpable at the time of laryngectomy and relatively so when, because of the extent and position of the primary lesion, one suspects the presence of or early development of cervical metastasis.

STEELE, Kansas City, Kan.

Nose

RECONSTRUCTION OF THE LACHRYMAL APPARATUS. F. L. ASHLEY, Plast. & Reconstruct. Surg. 17:64 (Jan.) 1956.

For reconstruction of the nasolachrymal apparatus, Ashley has used polyethylene tubing. The tubing is covered by a tubed skin graft with the raw surface outward, for lining the new tract. The natural flow of tears did not seem to be interfered with in his experience, and the results were otherwise also satisfactory. Drawings illustrate the technique, and there is a brief bibliography.

SELTZER, Philadelphia.

THE SURGICAL CLOSURE OF A LARGE ANTERIOR PERFORATION OF THE NASAL SEPTUM. S. CLIMO, Plast. & Reconstruct. Surg. 17:410 (May) 1956.

Climo presents a new technique for the surgical closure of a large anterior perforation of the nasal septum. There are three essential incisions: 1. A modified bilateral intercartilaginous incision that communicates with a transection of the columella, exposing the greater part of the inferior border of the septum. Enough skin is elevated over the bony- cartilaginous dorsum to allow a good view of the field. 2. A longitudinal incision along the floor of each nasal fossa to allow wide elevation of the mucoperiosteum and the mucoperichondrium on either side of the septum. 3. The most important incision is that made along the deep aspect of the upper side walls of the nose roughly parallel to and about ¼ in. from the dorsal border, carefully carried to the root of the nose. This permits the reflected portion of the septal membrane to be closed with the elevated septal mucoperichondrium. The upper lateral cartilages are freed from their attachment to the septal cartilage along the entire dorsum to allow direct vision. Two joined double-pedicled flaps are thus formed—upper and lower—of which the upper is most important. The individual membranous openings are closed with vertical mattress approximation sutures and an occasional through-and-through fixation suture so as to stagger the levels of the closed wound. Then the mucoperiosteum and mucoperichondrium are replaced on the body of the cartilage surfaces, and the nasal cavities are carefully packed with petrolatum (Vaseline) gauze. Finally, the columella is reattached to the lower border of the reconstructed septum. No stitches are needed for the intercartilaginous incision. A molded splint is used externally.

SELTZER, Philadelphia.

A SATISFACTORY NON-ADHERENT INTRANASAL PACK. G. H. GRAY and H. W. JONES JR., Plast. & Reconstruct. Surg. 17:471 (June) 1956.

Iodoform or petrolatum (Vaseline) gauze is said by the authors to be customarily used for postoperative nasal packing, and their removal is frequently accompanied by hemorrhage. To avoid this difficulty, the authors have made use of Telfa dressings for intranasal packing; they are made of polyester plastic film with an absorbent, nonwoven cotton backing. The film has about 200 very fine perforations per square inch, allowing drainage when the smooth surface is applied over the wound. In 918 human cases it has seldom adhered to any type of wound, though there has been occasional slight bleeding. Compared with the results from the use of petrolatum gauze, the result has been less bleeding, less injury to tissues, and less discomfort to the patient.

SELTZER, Philadelphia.

SURGICAL TREATMENT OF OZENA: THE USE OF A PROSTHESIS UNDER THE MUCOSA OF THE SEPTUM. LEON DEGELS, Acta oto-rhino-laryng. belg. 10:61, 1956.

The author discusses the problem of ozena and reviews the many attempts to narrow the patency of the nares. He mentions the various preparations that have been used in this

attempt and gives the reasons for their failure. He has employed a graft of the fibers of polyethylene, which he has formed in the shape of small pellets of various sizes. These he inserted under the mucous membrane of the septum in such a fashion to give an almost complete approximation between the septum and the inferior turbinates. He usually waits three months before operating on the other side of the septum. He has reported a series of 10 cases with excellent results in that there was a disappearance of the crusts and a free secretion of normal mucus between the inferior turbinates and the septum.

PERSKY, Philadelphia.

Pharynx

PERSISTENT SORE THROAT IN TONSILLECTOMIZED PERSONS. A. R. HOLLENDER, Eye, Ear, Nose & Throat Month. 35:117 (Feb.) 1956.

Hollender has analyzed a group of patients with "sore throat" persisting after removal of the tonsils. Tonsil tags or remnants accounted for over 40% of these cases. Causes for sore throats other than disease of the tonsils were found in a considerable number of patients. Among these causes may be listed active sinusitis with postnasal discharge, nasal deformities, nasal allergy, excessive smoking, excessive talking, and the inhalation of irritating fumes. Psychological factors may cause or complicate sore throats. Rational therapy requires control of the original etiologic factor. In those cases in which tonsil tags are present their removal is justified if it can be demonstrated that the persistent sore throat is due to the presence of these tonsillar remnants.

ABSTRACTER'S COMMENT: The commonest causes of persistent sore throat despite tonsillectomy are poor surgical technique and incorrect diagnosis. Success in surgical treatment is always dependent upon the technical skill and the diagnostic acumen of the surgeon. When the surgeon lacks either or both, the patient must depend upon luck. When his luck fails him, if he had persistent sore throats, he must continue to have his sore throats. This constitutes a challenge not just to otolaryngologists but to the medical profession as a group, for much of the tonsil surgery done in this country is done by men other than trained otolaryngologists.

JENNES, Waterbury, Conn.

Miscellaneous

CIGARETTE SMOKING, BRONCHIAL CARCINOMA AND CILIARY ACTION. A. C. HILDING, Ann. Otol. Rhin. & Laryng. 65:116 (March) 1956.

Hilding has shown experimentally that India ink and cigarette tar can be caused to accumulate for a matter of hours over areas of deciliated tracheobronchial mucosa.

Therefore, if such nonciliated islands of mucous membrane occur in the tracheobronchial tree of the smoker of tobacco, then the resulting accumulations of concentrated tar in such areas would duplicate closely the conditions of the experiments of carcinogenesis on the skin by means of tar.

The all-important question is are there such accumulations on naturally occurring squamous islands of the tracheobronchial mucosa? The author believes so, considering the known data. Possibly this is the key to carcinogenesis in the bronchial tree from any inhaled carcinogenic substance of whatever nature.

STEELE, Kansas City, Kan.

THE USE OF ANTIHISTAMINES IN ALLERGIC DISEASE. FREDERICK R. BROWN, New York J. Med. 56:2711 (Sept. 1) 1956.

This is another article in the series on "Fundamentals of Modern Allergy." Dr. Brown reports from the Allergy Clinic of Bellevue Hospital, Third Division. When allergen and allergic antibody combine, histamine or some similar substance is formed. To prevent allergic reactions, four methods are used: (1) elimination of or avoidance of allergens, (2) specific hyposensitization by allergenic extracts, (3) suppression of the action of histamine by antihistamines, and (4) inhibition of the allergic action through steroid therapy. The best results are in seasonal hay fever, especially in the edematous phase. Antihistamines are most effective in the beginning of the hay fever season. As time goes on, the symptoms become worse and relief is less easily obtained. The use of antihistamines is not a substitute for specific hyposensitization. But the use of antihistamines must be reevaluated for each individual patient. Some, such as Perazil and Phenergan, have a prolonged action.

Others have a delayed action, and so must be given over a longer period. Swallowing the tablets is the preferred method of administration. Dosage varies with each person, and the smallest amount consistent with effect should be administered. If no relief ensues within an hour, dosage should be increased. One may combine antihistamines with aminophylline or ephedrine in antiasthmatic preparations. Trial and error must be combined with common sense in working out a relief program. Antihistamines should be used to complement rather than substitute for immunologic therapy.

I. W. VOORHEES, New York.

ALLERGY TO PENICILLIN. SHEPPARD SIEGAL, New York J. Med. 56:2859 (Sept. 15) 1956.

Siegal, from the Adult Allergy Clinic of Mount Sinai Hospital, states that drug allergy has always been an annoying factor in medicine. Fortunately, most of the antibiotics give infrequent evidence of intolerance, but with ever-increasing varieties of these helpful agents we may well expect more adverse reports. The author sets down four types of reaction: T_1. I, the delayed, is the commonest, with an incubation period of from five days to eight weeks. It may simulate serum sickness, but urticaria is most frequently seen. Type II, accelerated and immediate, is uncommon and occurs chiefly in persons who have had penicillin months or weeks previously and therefore have been sensitized. The immediate type calls forth serum sickness, angioedema, asthma, and anaphylaxis. Type III, which includes hyperergic reactions, is rare, but these reactions are likely to be seen in the skin as various types of eruptions with occasional symptoms in the heart or kidneys which are reversible. Type IV, consisting of "id-like" or erythematovesicular reactions, is common and is secondary to Type I in incidence. These reactions appear hours or days after the administration of pencillin. Itching of the skin with papulovesiculation is seen, probably activated from an already existing sensitivity. Type V is a contact dermatitis, commonly seen in nurses and other attendants who handle ointments, etc.

Curiously, children seem to tolerate penicillin better than adults! If they react adversely, the sequence is usually minor, and in case of urgency, e.g., high fever, etc., one may continue with caution to administer the drug. The persistence of Type I reactions sometimes lasts a year.

Skin tests for penicillin sensitivity have limited value. Positive patch tests are helpful in diagnosing dermatitis incursion, but the instilled intracutaneous drop read 24 to 48 hours later is, perhaps, more trustworthy. Negative skin tests may be misleading and do not exclude complications. Desensitization of penicillin allergy is difficult and may be dangerous, but is useful in certain limited cases. The most feared reaction is, of course, anaphylaxis, which may prove to be fatal. It often occurs when the first penicillin administration is given following a long interval since the previous exposure. It does not occur with Salk vaccine, as has been thought, and is not due in any case to the procaine commonly associated with injections. The procaine is not the guilty agent, but the penicillin may be.

To reduce the hazards of giving penicillin the following precautions must be taken: One should not give it for every little disability, especially for "a cold," which is thought now to be due to a virus. Viral diseases seem immune to many antibiotics. A careful history will bring out previous incidents in a patient who has had penicillin months or weeks previously. The patient will remember what occurred. We must beware of former evidences of allergies, especially in bronchial asthma. Moreover, "penicillin shock often occurs in the absence of any observation of prior penicillin allergy." The site of injection is preferably the outer upper arm because, in case of urgency, a tourniquet can be applied proximally to shut off the immediate absorption. Injection into a blood vessel is always dangerous. It is unwise to give penicillin immediately after surgery, that is, "before the patient comes out." A fatality may result.

When untoward symptoms appear, epinephrine, 0.5 to 1 ml., should be given, and repeated as necessary. Shock may occur after taking an oral tablet or a troche or after instillation into the antrum. Protracted shock calls for whole blood or plasma intravenously.

ABSTRACTER'S COMMENT: This is an instructive and well-considered essay in the series concerning "Fundamentals of Modern Allergy," sponsored by the New York Allergy Society and written by its members.

I. W. VOORHEES, New York.

INHALED FOREIGN BODIES. C. L. N. ROBINSON and WILLIAM W. MUSHIN, Brit. M. J. 2:324 (Aug. 11) 1956.

From the Thoracic Center of the Sully Hospital, Glamorganshire, the authors report experiences with foreign bodies in the lower airways. A small foreign body will pass into the right bronchus, since this is the larger. A piece of meat or some other object may be of the size to fill the glottic chink, thus bringing complete asphyxia in a few minutes. The history is dramatic; there are coughing, dyspnea, and cyanosis. However, a very small foreign body may enter the posterior segmental bronchi and produce a minimum of symptoms. Thus in any case of wheezing, coughing, etc., in a child, foreign body should be suspected when atelectasis, diminished movement, dullness on percussion, decreased air entry, displaced apex beat, and a high diaphragm on the affected side are found. X-ray may be of little value if the foreign body casts no shadow. Anesthesia must provide the bronchoscopist with a tranquil oxygenated patient, with reduced reflexes. However, to abolish reflexes entirely may prove dangerous to life. Surface analgesia as a preliminary is helpful, and is sufficient in most adults, where "unconsciousness is rarely indicated." Children below 10 are put to sleep in bed by a basal narcotic such as rectal tribromoethanol (Bromethol), and carried to the operating room, where trichloroethylene and oxygen are administered. The mucous membrane is dulled by a small amount of local anesthetic, but there is danger from an overdose. A summary of 25 cases in well-tabulated form is presented in regard to age, sex, duration of symptoms, history and examination, x-ray findings, findings at bronchoscopy, and comment.

Prevention of foreign body intake is of primary importance, and is stressed by all who work in this field. Parents often set a bad example by holding pins and tacks in the mouth. "The adage is still true that if nothing except food was put in the mouth, the inhalation of a foreign body would be a rare accident." (This must be accepted with definite reservations, for an inhaled piece of meat in a normal adult while laughing at the table has been known to prove fatal.) In such a case the person dies very quickly from asphyxia. Nevertheless, it is true that "The majority of inhalation accidents may be prevented."

I. W. VOORHEES, New York.

News and Comment

ANNOUNCEMENTS

Postgraduate Course in Esophageal Speech and Organic Voice Problems.—The Third Postgraduate Course in Esophageal Speech and Organic Voice Problems, under the direction of Nathaniel M. Levin, M.D., F.A.C.S., and faculty, will be given from June 19 to June 30, 1957. The Course is sponsored by the University of Miami School of Medicine.

Traineeships (tuition and traveling expenses) valued at $250.00 are available on application. Otolaryngologists and speech pathologists are eligible.

For information write to Dean Homer Marsh, University of Miami School of Medicine, Coral Gables, Fla.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. G. M. T. Hazen, 208 Canada Building, Saskatoon, Sask.
Secretary: Dr. G. A. Henry, 328 Medical Arts Building, Toronto.
Place: Banff Springs Hotel. Time: June 17-19, 1957.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Bronchoesophagological Society

President: Dr. Theodor Hünermann, Düsseldorf.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth International Congress of Bronchoesophagology.
Place: Philadelphia. Time: May 12-13, 1957.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: 1958.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 90 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958 (afternoons only).

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Washington, D. C. Time: May 3, 1957.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Percy E. Ireland, 100 College St., Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.

SECTIONS:

Eastern.—Chairman: Dr. Joseph Sullivan, Toronto, Canada.
Southern.—Chairman: Dr. Mercer G. Lynch, New Orleans.
Middle.—Chairman: Dr. Paul M. Moore, Cleveland.
Western.—Chairman: Dr. Sigurd v. Christierson, San Francisco.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37.
Secretary-Treasurer: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Place: Statler Hotel, Washington, D. C. Time: May 4, 1957.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Joseph Gilbert.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

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Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago.

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Secretary-Treasurer: Major Stanley H. Bear, M. C., 3810th USAF Hospital, Maxwell AFB, Ala.

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Ref. Archives of Otolaryngology, February 1953

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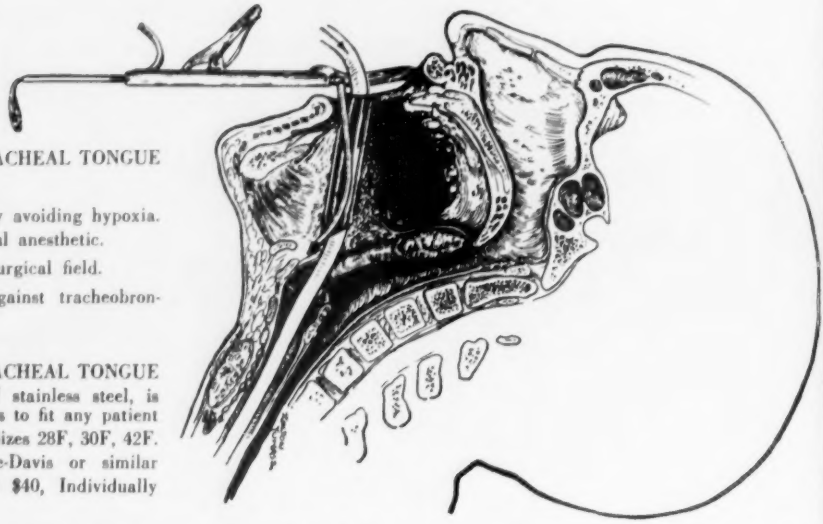

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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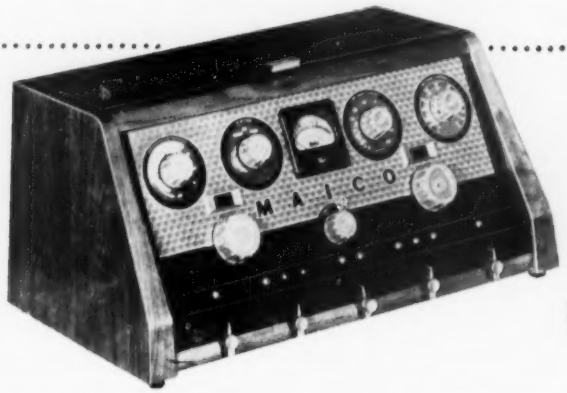

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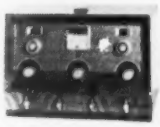

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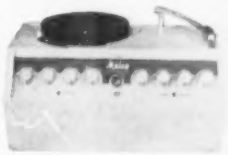

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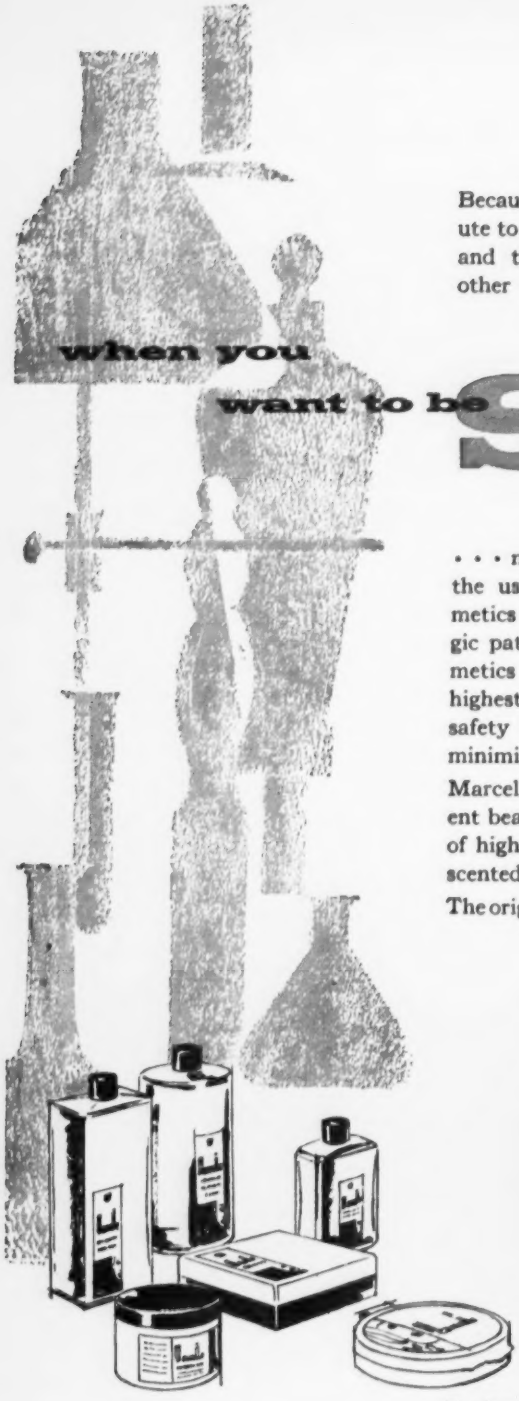

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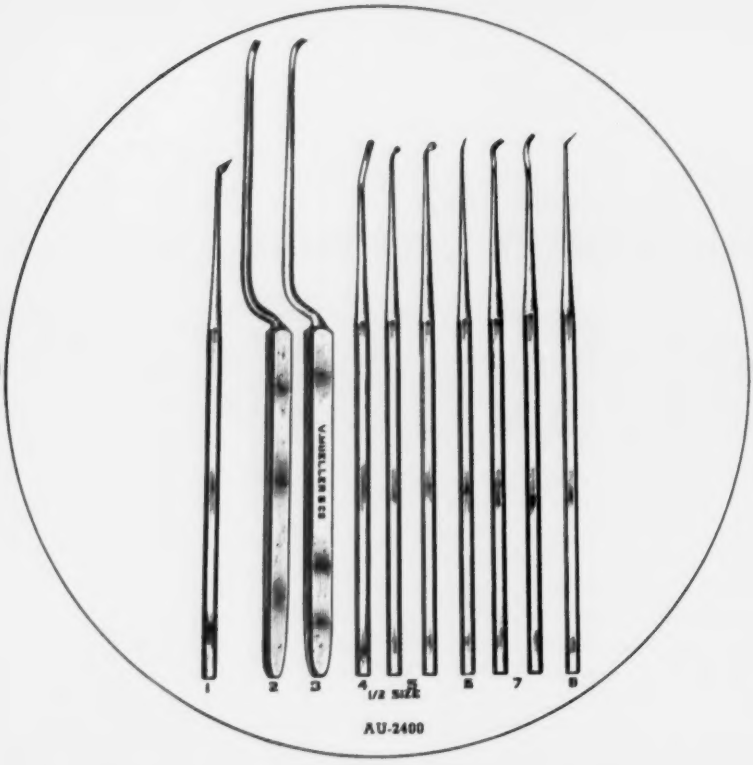

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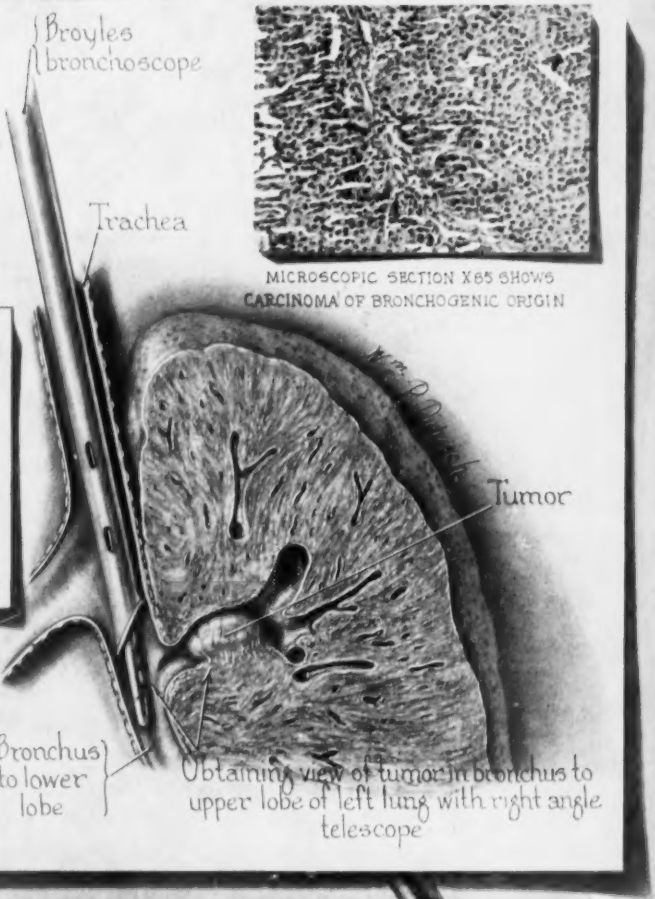
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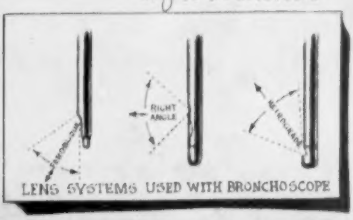

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